“A Comparative Study of Ropivacaine 0.5% and Ropivacaine 0.5% with Dexmedetomidine 50µg in Ultrasound Guided Supraclavicular Brachial plexus Block for Upper limb Orthopedic Surgery”.

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CERTIFICATE

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DECLARATION

I **Dr. H.M. HAJASHAREEF** solemnly declare that the dissertation titled **"A Comparative Study of Ropivacaine 0.5% and Ropivacaine 0.5% with Dexmedetomidine 50µg in Ultrasound Guided Supraclavicular Brachial plexus Block for Upper limb Orthopedic Surgery"** is a bonafide work done by me at Government Kilpauk Medical College under the guidance and supervision of **Prof.Dr.G.R.Rajashree,MD,DA,** Department of Anaesthesiology.

This dissertation is submitted to the Tamil Nadu Dr. M.G.R. Medical University towards the partial fulfillment of the requirements of M.D. Branch – X, Anaesthesiology degree examination.

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Date: 09.10.2014. **Dr. H.M. HAJASHAREEF**

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LIST OF ABBREVIATIONS

ASA	-	American society of anaesthesiologist
VAS	-	Visual analog scale
R	-	Ropivacaine
RD	-	Ropivacaine and Dexmedetomidine
MAC	-	monitored anesthesia care
µg	-	microgram
mg	-	milligram
ml	-	milliliter
ng	-	nanogram
C_m	-	Minimum blocking concentration
min	-	minutes
DOS	-	Duration of surgery
DOA	-	Duration of Analgesia
On. sen	-	onset of sensory block
On. mot	-	Onset of motor block
Do. Sen	-	Duration of sensory block
Do. Mot	-	Duration of motor
Res. An	-	Rescue Analgesia
Sed. Scores	-	Sedeation Scores
S/E	-	Side Effects

ABSTRACT

Background and objectives: Evaluated the effect of adding dexmeditomidine 50µg to ropivacaine 0.5% for supraclavicular brachial plexus block. The primary end points were the onset and the duration of sensory and motor block and duration of analgesia.

Methods: 50 ASA I and II patients scheduled for elective mid humerus, forearm, hand surgery were divided into 2 equal groups in a randomized double blind fashion. In group R (n=25), 30ml of 0.5% ropivacaine +1 ml saline and in group RD (n=25), 30ml of 0.5% ropivacaine +1ml (½ml of dexmeditomidine 50µg mix with 1/2ml of saline) were given. Onset time of sensory and motor block, duration of sensory and motor block and duration of analgesia were recorded.

Results: Demographic parameters were comparable in both groups. Onset time of Sensory and motor block were shorter in group RD than in group R (P=0.001). Duration of Sensory and motor blockade were longer in group RD than in group R (P=0.001). Duration of analgesia was longer in group RD than in group R (P=0.001). Systolic blood pressure levels in group RD at 15, 30, 45, 60, 90, 120, 180, 240, 300 minutes were significantly lower than those in group R (P=0.001). Diastolic blood pressure levels in group RD at 15, 30, 45, 60, 90, 120, 180, 240, 300 minutes were significantly lower than those in group R (P=0.001). Heart

rate levels in group RD except basal measurements were significantly lower than those in group R (P=0.001).

Conclusions: Dexmedetomidine when added to ropivacaine for brachial plexus block shortens the onset time and prolongs the duration of the block and the duration of postoperative analgesia.

Key words: Dexmedetomidine, Ropivacaine, Ultrasound,

Supraclavicular Brachial Plexus Block

TABLE OF CONTENTS

INTRODUCTION[23, 32.]

Brachial plexus blockade by supraclavicular approach is rapid onset and complete and predictable anaesthesia for mid humerus, forearm and hand surgery. This approach is also known as spinal anaesthesia of the upper limb because of its common application for upper limb surgical procedures. The anatomic characteristics are the key factor its high success rate. The compact structure of the plexus is an added advantage to nerve block at this level.

Peripheral nerve blocks provide good operating conditions when it used optimally. They not only provide excellent intra operative anaesthesia but also good post-operative analgesia. They cause the least interference with the vital physiological functions of the body, reduction in stress response, systemic analgesia requirements, avoiding polypharmacy, opioid related side effects and general anaesthesia requirements.

Regional blocks have traditionally been performed by eliciting paraesthesia, anatomical landmarks and fascia clicks. Serious complication like pneumothorax occurs due to blind technique because it is mainly dependent on anatomical landmarks. Over the last two decades Neuro stimulation was the Gold standard technique for nerve identification in regional blocks. But it does not ensure the required level of nerve block. It also causes damages to the nerve structures by direct puncture.

Ultrasound visualisation of anatomical structures facilitates safe methods for regional blocks. This technique enables the anaesthetist to secure

an optimal needle positioning and to monitor the distribution of local anaesthetics in real time. The amount of local anaesthetic required for effective nerve block can be minimised by directly monitoring its distribution. Local anaesthetic blocks the nerve but is not blocked by the needle.

Most of the local anaesthetic agents developed in the first half of 20th century (1900-1940) were basically amino ester compounds. They lost their importance due to their shorter duration of action and associated allergic reactions and systemic side effects. This paved the way for synthesis of newer agents namely the amino amide compounds such as bupivacaine, levobupivacaine, ropivacaine etc.

Ropivacaine is considered to be superior over bupivacaine, as it provides more differential block when given via epidural route. Motor block is not preferable during epidural labour and postoperative analgesia. In these situations ropivacaine offers greater sensory and motor separation. Ropivacaine is less cardiovascular and central nervous system toxicity than bupivacaine. The decreased systemic toxicity is better when a potential for high concentrations of local anaesthetic agents are used in peripheral nerve block and epidural anaesthesia. Because of its advantages, ropivacaine may be a better choice to bupivacaine.

To prolong the duration of analgesia various drugs have been studied as adjuvants to local anaesthetic solution and techniques such as the continuous catheter placement in the brachial plexus have evolved. These

adjuvant drugs added to peripheral nerve block are expected to enhance the duration of analgesia without causing any systemic adverse effects and prolonging motor blockade. Novel α-2 adrenergic agent, Dexmeditomidine is eight times more selective for α-2 adrenoreceptor than clonidine. It has an analgesic, sedative and good cardiovascular stabilizing effect.

The present study was designed to compare between ropivacaine and ropivacaine with dexmeditomidine in ultrasound guidance supraclavicular brachial plexus block scheduled for upper limb orthopaedic surgery.

HISTORY[21]

Regional anesthesia traces its origin to Dr. Karl Koller a young Austrian ophthalmologist, who in 1884 instilled a cocaine solution into his own eye, tested its effectiveness and employed for topical corneal anesthesia in patients undergoing eye surgeries.

The first brachial plexus block was performed by William Halsted in1889. He directly exposed the brachial plexus in the neck to perform the block and he used cocaine.

Harvey Cushing, a Halsted's surgical residents who applied cocaine to the brachial plexus during a forequarter amputation for sarcoma in the year 1900.

German surgeon named Diedrich Kulenkampff performed the first percutaneous supraclavicular block in 1911. He subjected himself to the supraclavicular brachial block.

Percutaneous approach to the brachial plexus from the axilla was first described by Georg Hirschel.

Kulenkampff first described classical supraclavicular approach to brachial plexus. With numerous blocks performed by Kulenkampff and his colleuge Persky and published their experiences without apparent major complications.

Continuous brachial plexus block technique was first described by F. Paul Ansbro in 1946. He followed a method of securing a needle in the supraclavicular fossa where he attached a tube connected with a syringe through which local anesthetic drugs are injected. The axillary approach was first performed by Accardo and Adriano in 1949.

The subclavian perivascular method was first described by Winnie and Collins in 1964 and it became popular due to its decreased risk of complication like pneumothorax when compared to the traditional Kulenkampff approach.

Raj first developed the infraclavicular approach. A technique for continuous brachial plexus block by using an intravenous catheter in the axilla was described by Selander in 1977.

The use of ultrasound for nerve blocks was first reported by P. La Grange and his colleagues in 1978.He performed supraclavicular brachial plexus blocks with the help of a Doppler blood flow detector and reported high success rate without complications.

B - Mode ultrasonography was used to demonstrate the anatomical structure of axilla and spread of local anesthetics in axillary brachial plexus block was reported by P. Ting and V. Sivagnanaratnam in 1989.

Stephan Kapral et al in 1994 published the first reported use of direct sonographic visualization for regional anesthesia. Over the past ten years however dramatic progress has been made.

AIM OF THE STUDY

The aim of the present study was to evaluate the clinical effectiveness, block quality and hemodynamic effects when addition of Dexmedetomidine 50µg to 0.5% Ropivacaine in ultrasound supraclavicular brachial plexus block. The following parameters were observed.

- Onset of sensory and motor blockade.
- Duration of sensory and motor blockade.
- Duration of Analgesia.
- Hemodynamic changes.
- Sedation.
- Time of Rescue analgesia.
- Complications.

BRACHIAL PLEXUS [28, 29, 30]

Brachial plexus block is one of the most commonly used peripheral nerve blocks in clinical practice. Hence the knowledge of the brachial plexus and of its distribution is absolutely essential for the effective blockade. Mastery of various techniques of brachial plexus anesthesia purely depends on familiarity with the vascular, muscular and fascial relationship of the brachial plexus throughout its formation and distribution.

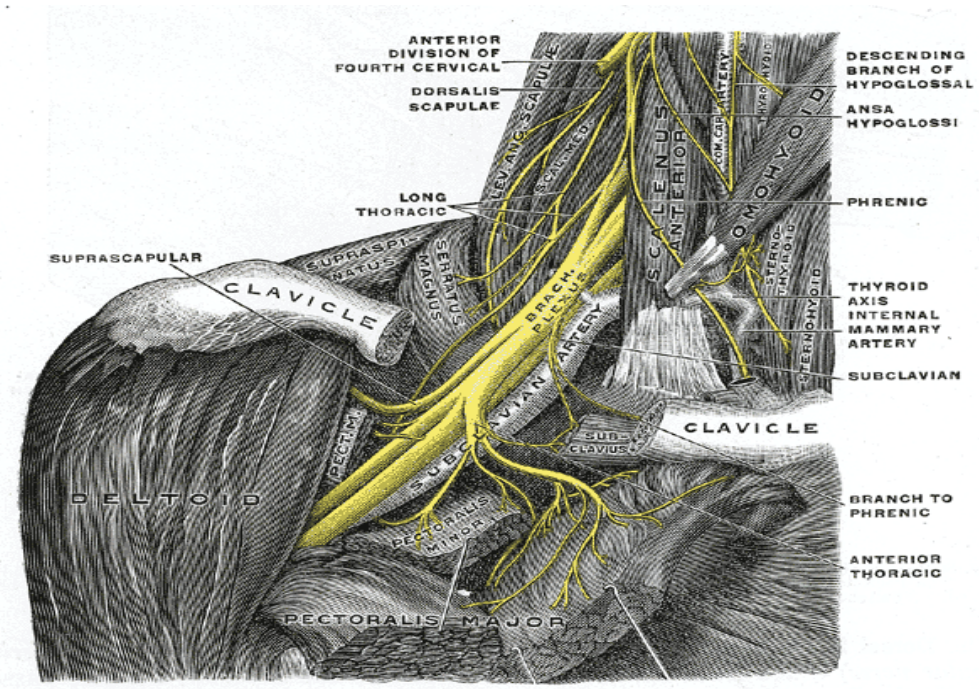
In its course from the intervertebral foramina to the upper arm, the fibers that constitute the plexus are composed consecutively of roots, trunks, cords, divisions and terminal nerves which are formed through a complex process of combining, dividing, recombining and finally re-dividing.

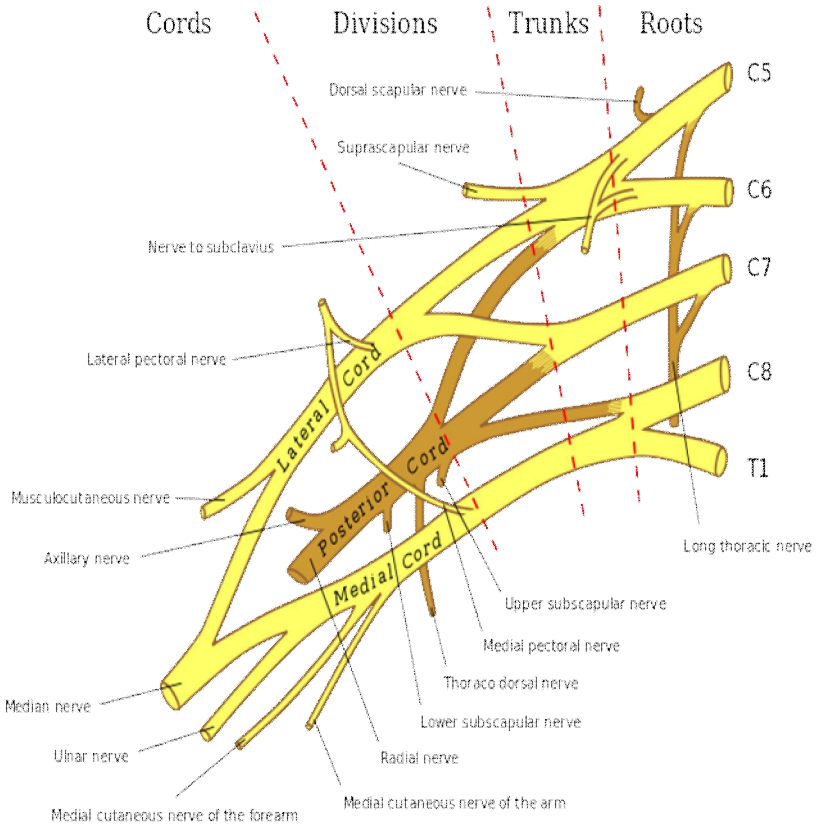
Formation of the plexus

Roots

The brachial plexus is formed by the union of anterior primary rami of fifth to eighth cervical nerve and first thoracic nerve with occasional contributions from the fourth cervical nerve (prefixed) above and second thoracic nerve (post fixed) below. These nerves unite to form trunks, which lie in the neck above the clavicle. The discrepancies are commonly related with the occurrence of a cervical rib, anomalous of first rib.

Brachial plexus




Cords
Divisions
Trunks
Roots
C5
C6
C7
C8
T1
Dorsal scapular nerve
Suprascapular nerve
Nerve to subclavius
Lateral pectoral nerve
Musculocutaneous nerve
Axillary nerve
Median nerve
Ulnar nerve
Medial cutaneous nerve of the forearm
Lateral Cord
Posterior Cord
Medial Cord
Long thoracic nerve
Upper subscapular nerve
Medial pectoral nerve
Thoraco dorsal nerve
Lower subscapular nerve
Radial nerve
Medial cutaneous nerve of the arm

Trunks

The emergence of five roots of the brachial plexus is from the intervertebral foramina. They lie in the gutter between the anterior and posterior tubercles of the corresponding transverse process. All five roots then become sandwiched between scalenus anterior and scalenus medius. Here the roots of C_5 and C_6 unite into the upper trunk, the root of C_7 continues as the middle trunk and those of C_8 and T_1 into the lower trunk. Each trunk divides behind the clavicle, into anterior and posterior divisions which unite in the axilla to form cords.

Cords

Three cords are formed when the six divisions, stream into the axilla and joint together. These cords are

- Lateral
- Medial
- Posterior

The union of the anterior divisions of the upper and middle trunks forms the lateral cord. The medial cord represents the continuation of the anterior division of the lower trunk. The posterior cord comprises of the posterior divisions of all the three trunks.

The composition of brachial plexus can be summarized as follows:

1. Five roots (between the scalene muscles) - the anterior primary rami of C_5 - C_8 and T_1

2. Three trunks (in the posterior triangle)

   a) Upper trunk - C_5 and C_6

   b) Middle trunk - C_7 alone

   c) Lower trunk - C_8 and T_1

3. Six divisions (behind the clavicle) - each trunk divides into an anterior and posterior division.

4. Three cords (within the axilla)

   a) Lateral cord-the fused anterior division of the upper and middle trunks C_5-C_7.

   b) Medial cord - the anterior division of the lower trunk C_8-T_1

   c) Posterior cord- formed by the union of the posterior divisions of all three trunks $C_5 – T_1$.

Branches

Branches are given off from roots, trunks, and cords.

1. Branches from the roots:
    a) Nerve to the serratus anterior (C5, C6, and C7)
    b) Muscular branches to
        - Longus cervices (C5-C8)
        - Scalene muscles (C5-C8)
        - Nerve to rhomboids (C5)
        - Scalene muscles (C_5-C_8)
        - Contribution to phrenic nerve
2. Branches from the trunks:
    a) Suprascapular nerve (C5-C6)
    b) Nerve to subclavius (C5-C6)
3. Branches from the cords:
    a) Lateral cord
        - Lateral pectoral nerve (C5-C7)
        - Lateral head of median nerve (C5-C7)

- Musculocutaneous nerve (C5-C7)

b) Medial cord

- Medial pectoral nerve (C8-T1)
- Medial head of median nerve (C8-T1)
- Medial Cutaneous nerve of arm (C8-T1)
- Medial cutaneous nerve of forearm (C8-T1)
- Ulnar nerve (C7, C8-T1)

c) Posterior cord

- Upper subscapular nerve (C5- C6)
- Lower subscapular nerve (C5-C6)
- Nerve to latissimus dorsi (C6, C7, C8)
- Axillary nerve (C5-C6)
- Radial nerve (C5, C6, C7, C8 T1)

Relations of the brachial plexus

Its roots pass through the fascia enclosed space between the scalenus anterior and the scalenus medius accompanied by subclavian artery and

invaginate the scalene fascia to form a neurovascular bundle. This fascia becomes axillary sheath in the axilla.

Trunks

The trunks of the plexus are enclosed in a sheath of prevertebral fascia in the posterior triangle. These trunks of brachial plexus are placed superficially and it is covered by the skin, platysma and deep fascia.

The upper and middle trunks when they stream across the first rib subclavian artery runs above, whereas the lower trunk which found behind the artery immediately posterior to the subclavian groove, may ridge the rib.

Division

The trunks splits into divisions at the lateral border of first rib which are present behind the three structures namely clavicle, the suprascapular vessels and subclavius muscle.

Cords

At the apex of axilla the cords are moulded and they segregate around the axillary artery.

The interscalene sheath

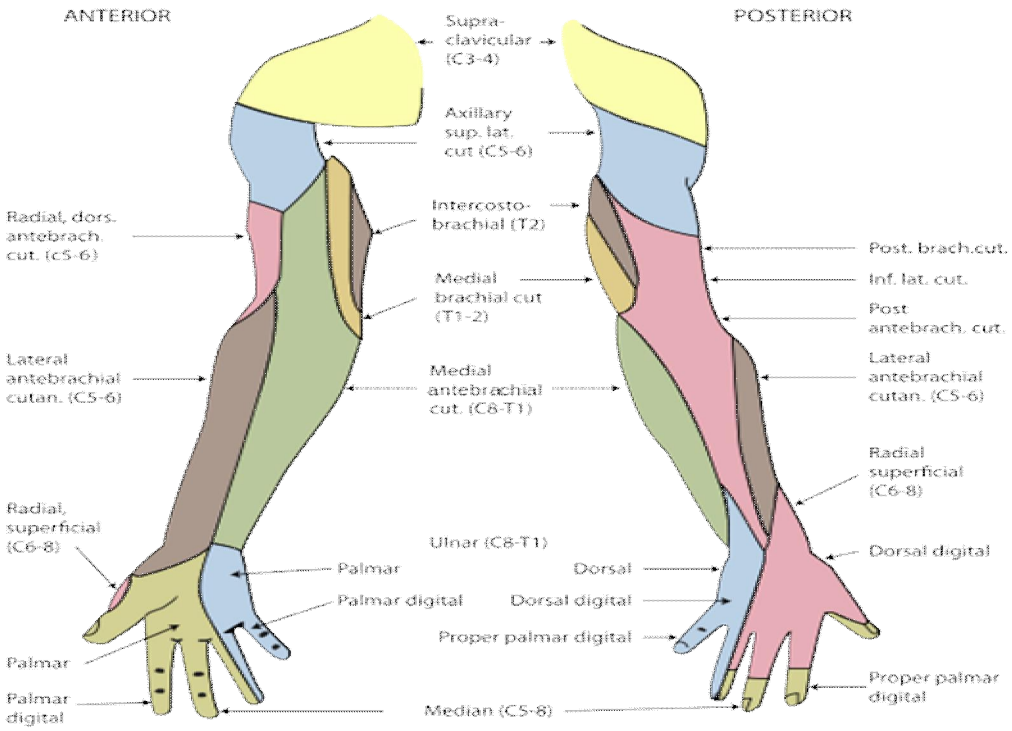
Roots of the brachial plexus originate in a trench between the two tubercles of the transverse process of the cervical vertebrae and situated in

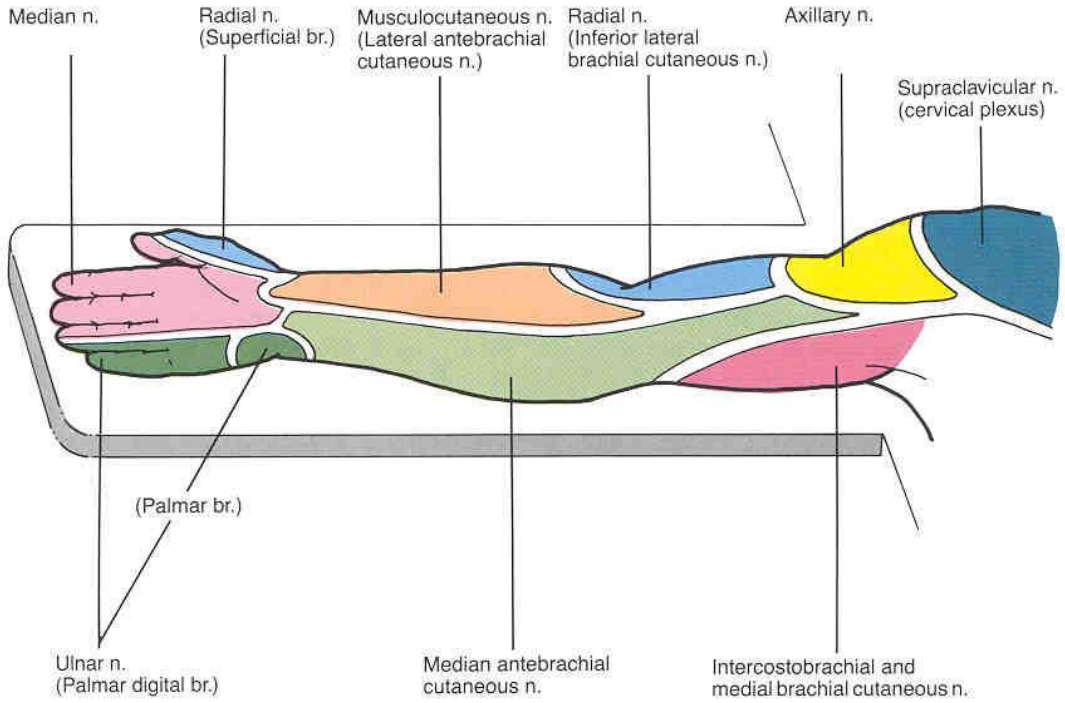
between two strands of fibrous fatty tissue space. Anterior and Posterior part of the sheath arises from the corresponding tubercles of cervical vertebrae. The front of the scalenus medius is covered by posterior part of sheath whereas posterior part of scalenus anterior is covered by anterior part of sheath.

Importance to Anesthetist

The interscalene sheath surrounding the brachial plexus along which local anesthetic solution may be injected to achieve brachial plexus block. It can be performed by interscalene or axillary approach.

Brachial Plexus - Branches





Anatomic considerations

The prevertebral and scalene fascia surrounds the brachial plexus. Fascial sheath constitutes prevertebral fascia and scalene fascia which extends from the intervertebral foramina to the upper arm. The existence of the facial sheath renders brachial plexus block easier. The sheath can be injected at any anatomical level that allows easy distribution of local anesthetic drugs and subsequent blockade of brachial plexus.

Brachial plexus blockade offers anesthesia of upper extremity surgical procedures. Brachial plexus are blocked by four techniques. They are

- Interscalene
- Supraclavicular
- Infraclavicular
- Axillary approach

The technique to be used in any case should be determined on the basis of surgical site, the required level of anesthesia, physical status and habitus of the patient.

Each technique has a chance of missing a nerve distribution that needs additional supplementation. The upper median aspect of the arm is not anaesthetised by any brachial plexus technique. This area innervated by the

medial brachial cutaneous nerve (C_8-T_1) and intercostobrachial nerve (T_2) which are not a part of the brachial plexus sheath. This nerve can be blocked by subcutaneous infiltration across the upper medial aspect of the arm using 3-5ml of local anesthesia solution to prevent tourniquet pain.

A part of the anterior shoulder is innervated by the superficial cervical plexus (C_1-C_4). This is effectively blocked by subcutaneous infiltration along the posterior border of the sternocleomastoid during a shoulder surgery.

Brachial plexus can be blocked at the level of roots, trunks, cords or peripheral branches. The block at each level has a distinct distribution of anesthesia, advantages, disadvantages and complications.

APPLIED PHYSIOLOGY[24]

Physiology of Nerve Conduction[32]

Neurons are the basic building blocks of the nervous system that respond to various stimuli. Integration and transmission of nerve impulses are specialized functions of neurons.

All peripheral nerves are elongated axons of neurons situated centrally. A typical peripheral nerve consists of bundles of motor, sensory and other fibres enclosed in the outermost covering called epineurium. Inside the epineurium, the perineurium surrounds the collection of bundles. Each bundle is surrounded by an endoneurium. Each nerve fibre in a bundle in layer of neurilemma or the axonal membrane. Depending on the presence or absence of myelin sheath, it can be a myelinated nerve fibre or unmyelinated nerve fibre.

The axonal membrane itself is made up of a bimolecular lipid palisade, interspersed with large protein molecules. The membrane lipids are largely phospholipids composed of a polar head group and a non-polar hydrocarbon tail.

The primary function of the cell membrane is to separate the extracellular from the intracellular environment. The major difference between these two environments is the ionic concentration. This disequilibrium provides the means for impulse conduction.

The most important ions in the respect are sodium and potassium. A membrane bound protein sodium potassium ATPase maintains normal resting equilibrium potential between - 50 mv to - 90 mv by pumping sodium ions out of the cell and potassium ions into the cell. A positive ion gradient from inside the membrane to the outside causes electro negativity inside the membrane. During nerve conduction the following changes occur in the cell membrane.

Resting phase

There is a potential difference across the membrane inside is negative, due to a higher concentration of Na^+ outside than the inside the cell. K^+ moves out of the cells and Na^+ moves in but because of more K^+ channels opened at rest, K^+ permeability is greater than Na^+ permeability. Therefore K^+ channels maintain the resting membrane potential.

Depolarization phase

During excitation, Na^+ channels in the cell membrane open briefly allowing Na^+ to flow into the cell, thereby depolarizing the membrane.

Repolarization phase

During this phase, opening of voltage gated K^+ channels occurs, results in passing of K^+ to the cell to restore electrical neutrality.

Restoration phase

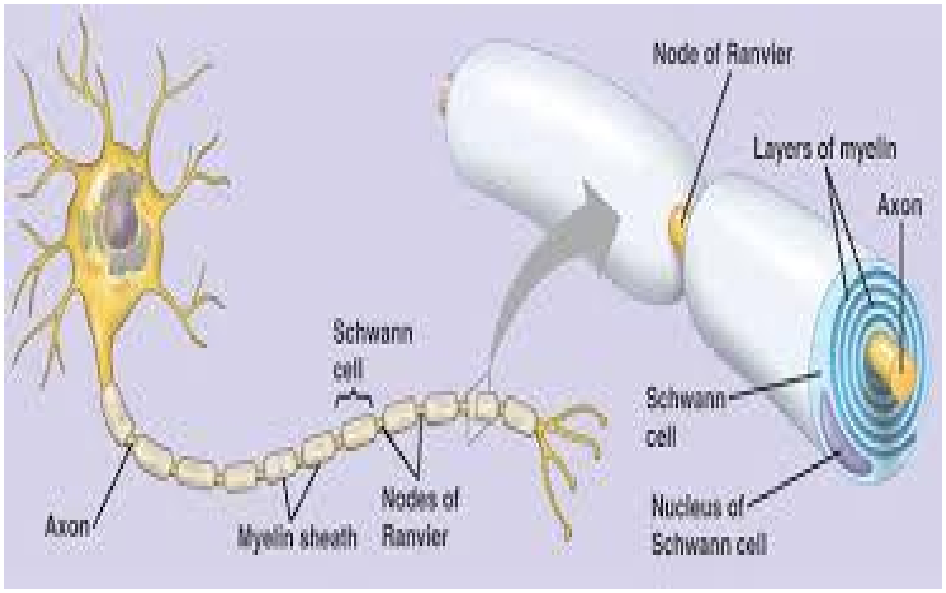
During this phase, Na^+ returns to the outside and K^+ re-enter the cell.

Distribution of ion channels in myelinated neurons

Voltage gated Na^+ channels are highly concentrated in the Nodes of Ranvier and the initial segment in the myelinated neurons. The initial segment and in sensory neurons, the first node of Ranvier are the sites where the impulses are normally generated and the other nodes of Ranvier are the sites to which the impulses jump during salutatory conduction.

The Na^+ channel is believed to be an integral membrane spanning protein. The three dimensional configuration of the proteins forms a pore through the neuronal membrane. Depolarization of the cell induces a configurational change on the Na^+ channel which causes it to open and allow ion passage. In many myelinated neurons, the Na^+ channels are flanked by K^+ channels that are involved in repolarization.

Nodes of Ranvier



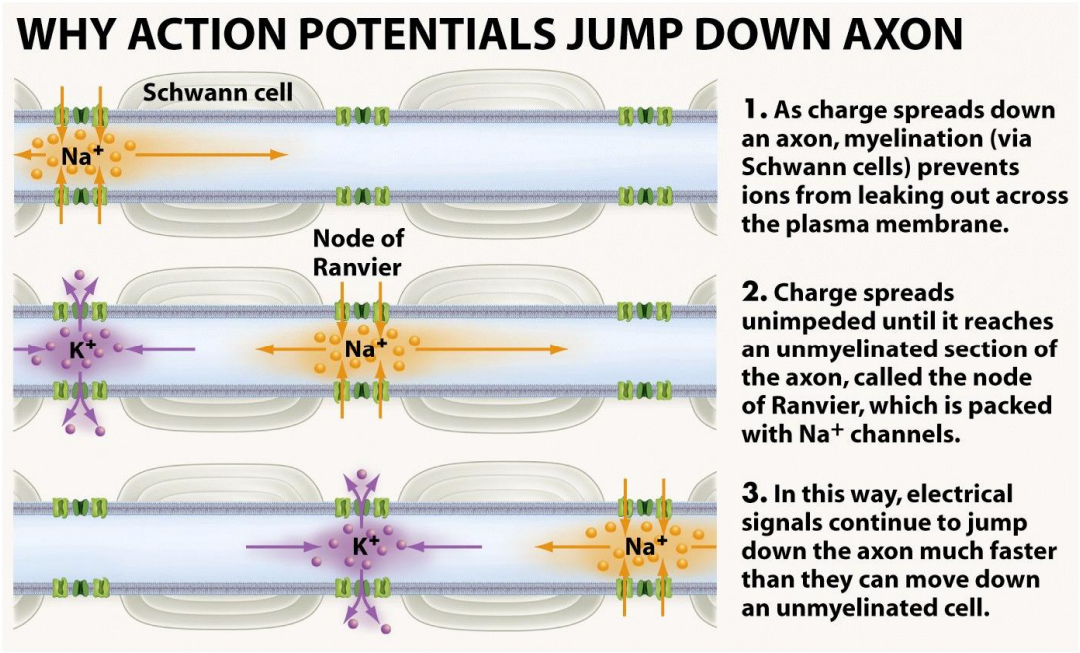


Figure 45-12b Biological Science, 2/e

Nerve Fibers[21]

Nerve fibers are classified into A, B, and C fibers. A- Fibers is further divided into α, β, γ and δ fibers. A- Fibers are myelinated somatic nerves, B - Fibers are myelinated preganglionic autonomic nerves and C - Fibers are unmyelinated nerves. The susceptibility of nerves to local anesthetics depends on their caliber, degree of myelination, speed of conduction.

Characteristics of different categories of nerve fiber

Characteristics	**Aα**	**Aβ**	**Aγ**	**Aδ**	**B**	**C**
Diameter (μm)	12-20	5-12	5-12	1-4	1-3	0.5-1
Conduction Speed	70-120	30-70	30-70	12-30	14-8	12
Myelination	+++	++	++	+	+	-
Function	Motor	Pressure touch	Proprioception	Pain temp	Vaso constriction	Pain temp.
Onset of block	5th	4th	3rd	2nd	1st	2nd

Differential nerve block

Within a single peripheral nerve, the complete block of pain fibers (Aδ and C) and sparing of motor and touch (Aα and Aβ) is known as differential block. This is may be due to

1. Time taken for a drug to diffuse into and along the course of nerve to affect various fibers.

2. The presence or absence of a myelin sheath may affect local anesthetic activity and penetration.

3. Not all axons have the same sensitivity to local anesthetic agents because of variations in Na^+ channel and membrane lipid content.

Nerve Penetration

The fibers innervating the distal portions of a limb are in the center of the nerve trunk. The more proximal structures are supplied from the outer layers of the trunk. Following deposition of the drug, the more proximal limb structures anesthetized before the distal ones.

- Proximal - early block
- Distal - delayed block

Regression of the block is primarily dependent on diffusion from the nerve and absorption into the local vasculature. Drugs with high lipophilic solubility diffuse slowly from local tissues.

Minimum blocking concentration (C_m)

It is the lowest concentration of local anesthetic agent that will block conduction in a nerve in vitro. In vivo, the drug is injected in and about nerve trunks, fibrous sheaths, fatty tissue and blood vessels. Therefore, before reaching the nerve, it is subject to dilution, dispersion, fixation, destruction and systemic absorption. Under these conditions, the minimum concentration necessary to block a nerve is much greater than the C_m.

Local anesthetics action on nerve fibers[24]

The primary action of local anesthetics on the nerves is electrical stabilization. The large transient increase in permeability to Na^+ necessary for propagation of the nerve impulse is prevented. Thus the resting membrane potential is maintained and depolarization in response to stimulation is inhibited.

Local anesthetics block sodium conductance by

- By action of local anesthetic drugs to sites on voltage gated Na^+ channels limits opening of the channels by inhibiting the conformational changes that underlie channel activation.

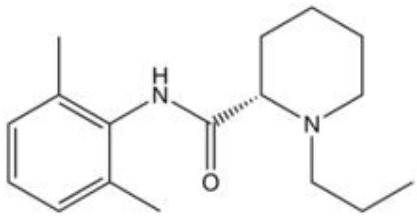
- Local anesthetic drugs produce nonspecific membrane expansion. There is an unfolding of membrane protein together with a disordering of the lipid component of the cell membrane with consequent obstruction of the Na^+ channel.

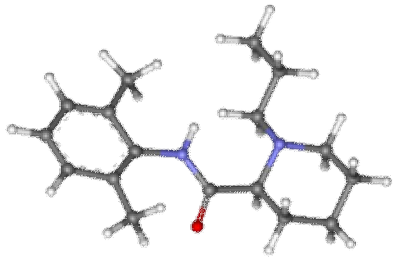
ROPIVACAINE HYDROCHLORIDE[25, 26, 27]

Ropivacaine belongs to a long-acting amide local anaesthetic drug. It was first synthesized as a pure enantiomer. When compared to bupivacaine it is less lipophilic with a less chance to penetrate motor fibers which is large and myelinated, hence it results in a decreased motor blockade. Because of less lipophilic it leads to less cardiovascular and central nervous system side effects than bupivacaine. The decreased systemic toxicity is better when a potential for high concentrations of local anaesthetic agents are used in peripheral nerve block and epidural anaesthesia.

Ropivacaine is considered to be superior over bupivacaine, as it provides more differential block when given via epidural route. Motor block is not preferable during epidural labour and postoperative analgesia. In these situations ropivacaine offers greater sensory and motor separation.

It belongs to mepivacaine family. It is introduced into the market during the year 1996.





Chemical

It is an amide local anaesthetic (*S*)-*N*-(2, 6-dimethylphenyl)-1-propylpiperidine-2-carboxamide.

It is a pure S (-) enantiomer.

It belongs to the pipecoloxylidide group of local anaesthetics.

It has a propyl group on the piperidine nitrogen atom. But bupivacaine has a butyl group.

Presentation

As a clear, containing racemic ropivacaine hydrochloride monohydrate (S - and R - enantiomers) in concentration of 0.2/0.5/0.75/1% equivalent to 2mg,5mg, 7.5 mg, 10mg/ml respectively. The preparation also contains sodium hydroxide equivalent to 3.7mg of sodium per ml.

Physiochemical properties

Molecular weight : 274

Pka : 8.1

Protein binding : 94%

V_D : 52-66 l

Elimination T ½ : 59-173 minutes

Clearance : 0.44 – 0.82l/min

Excretion : Renal 86%

Routes of administration/ doses

Ropivacaine may be administered topically, by infiltration, epidurally. Maximum recommended dose of ropivacaine is 3mg/kg. Alkalinization of 0.75% ropivacaine leads to prolongation of analgesia.

Indication in adults	**Concentration (%)**	**Volume**	**dose**
Surgical anaesthesia Lumbar epidural (caesarean section)	0.75	15-20 ml	113-150 mg
Lumbar epidural (other surgery)	0.75	15-25 ml	150-200 mg
	1	15-20 ml	150-200 mg
Thoracic (single block for postoperative pain relief)	0.75	5-15 ml	38-113 mg
Intrathecal administration	0.5	3-4 ml	15-20 mg
Peripheral administration	0.75	10-40 ml	75-300 mg
Field block	0.75	1-30 ml	7.5-225 mg
Post-operative pain Lumbar epidural (continuous infusion)	0.2	6-10 ml/h	12-20 mg/h
Thoracic epidural (continuous infusion)	0.2	6-14 ml/h	12-28 mg/h
Peripheralnerveblock (continuous infusion)	0.2	5-10 ml/h	10-20 mg/h

Field block	0.2	1-100 ml/h	2-200 mg
Intra-articular injection	0.75	20 ml	150 mg
Labour pain (Lumbar epidural)	0.75		
Bolus	0.2	10-20 ml/h	20-40 mg
Intermittent top-ups	0.2	10-15 ml/h	20-30 mg
Continuous infusion	0.2	6-14 ml/h	12-28 mg/h
In children - Caudal epidural block (below T12)	0.2	1 ml/kg	2 mg/kg
Peripheral nerve block (eg. Ilioinguinal block)	0.5	0.6 ml/kg	3 mg/kg

Mechanism of action

Local anaesthetics block the generation and conduction of nerve impulses presumably by increasing the threshold for electrical excitation in the nerve, by slowing the propagation of the nerve impulse, and by reducing the rate of rise of the action potential.

In general the progression of anaesthesia is related to the diameter, myelination and conduction velocity of nerve fibres. Clinically the order of loss of nerve function is as follows.

1. Pain

2. Temperature

3. Touch

4. Proprioception

5. Skeletal muscle tone

Ropivacaine leads to reversible inhibition of sodium ion influx. Hence it produces conduction blockade in nerve fibres. This is potentiated by potassium channels inhibition. Because of its less lipophilicity and less likely to penetrate large motor myelinated fibres. It has differential block due to its selective action on the nerve fibers.

S-ropivacaine is more potent and less cardio toxic than R-ropivacaine.

Pharmacodynamics

Central nervous system

The principle effect of ropivacaine is reversible neural blockade. This leads to a characteristically biphasic effect in the central nervous system. During accidental over dosage or direct vascular injections the clinical signs are numbness of tongue, light headedness, visual and auditory disturbances, muscular twitching's, tremors. When plasma level continues to raise central nervous system excitation is rapidly superseded by depression, drowsiness, disorientation and coma.

Ropivacaine is less lipophilic than bupivacaine. Because its stereo selectivity, it has higher threshold for cardio toxicity and Central nervous system toxicity. The Central nervous system effects occurred earlier than cardio toxic symptom.

Cardiovascular system

Ropivacaine is less cardio toxic than bupivacaine. In toxic concentrations the drug decreases the peripheral vascular resistance and myocardial contractility thereby producing hypotension and possibly cardiovascular collapse. It has biphasic vascular effect causing vasoconstriction at low but not at high concentrations.

Other effects

Ropivacaine does not compromise the uteroplacental circulation.

Ropivacaine inhibits platelet aggregation.

It has antibacterial activity in vitro.

Pharmacokinetics

The absorption of local anaesthetic agents is related to

- The site of injection

(Intercostal>caudal>epidural>brachial plexus>subcutaneous)

- The dose – a linear relationship exists between the total dose and the peak blood concentrations achieved.
- The presence of vasoconstrictors which delay the absorption.

Distribution

Ropivacaine is 94% protein bound and predominantly binds to α-1 acid glycoprotein. The volume of distribution is 52-60 litres. It has biphasic absorption profile from epidural space with half-life of 4 hours and 14 minutes in adults.

Plasma concentration of ropivacaine mainly depends on the following factors.

1. The total dose administered and the route of administration.

2. Haemodynamic and circulatory condition of the patient.

3. Vascularity of the administration site.

During continuous epidural infusion of ropivacaine the total plasma concentration increases due to an increase in the protein binding and decrease in the clearance of ropivacaine.

During epidural administration for caesarean section, it rapidly crosses the placenta which results in the equilibrium between the maternal and foetal circulation. Due to increased concentration of α1 - acid glycoprotein in

maternal side than in foetal side, the amount of drug present in the maternal circulation is more than the foetal circulation.

Metabolism

In the liver ropivacaine is metabolised by aromatic hydroxylation via cytochrome P450 (CYP) 1A2 to 3′ - hydroxyl - ropivacaine which is the major metabolite. The isoenzyme CYP3A4 is also involved in the metabolism of ropivacaine to 2′, 6′-pipecoloxylidide.

Excretion

Ropivacaine is mainly excreted via kidney, which is accounting for 86%, 37% of 3 – hydroxyl – ropivacaine is excreted in the urine and 1% is unchanged. Terminal half-life is 59 – 173 minutes.

The clearance is 0.44 – 0.82 litres per minute.

Due to biphasic absorption, the elimination half – life is longer after epidural administration (approximately 4.2 hours) than after intravenous administration.

Side effects

Allergic responses to the amide-type of local anaesthetic agents are extremely rare. The side effects are predominantly due to excessive plasma concentrations of the drug. The common side effects are hypotension, nausea, vomiting, bradycardia and headache.

Higher incidence of cardiovascular toxicity like bradycardia, hypotension reported in

- Age more than 60 years
- sudden IV injection
- Massive absorption from peripheral nerve blocks.

Drug interactions

Due to toxic effects of additive action, care to be taken with patients receiving other amide – type local anaesthetics.

Co-administration of CYP1A2 inhibitors like fluvoxamine with ropivacaine results in increased plasma concentration of ropivacaine. Other drugs like theophylline and imipramine may also metabolised by CYP1A2 via competitive inhibition. Hence careful administration of these drugs is needed due to its possible interaction.

Special points

The onset and duration of conduction blockade is mainly related to

- pKa
- Lipid solubility
- Extent of protein binding

A low pKa and high lipid solubility are associated with a rapid onset time.

High degree of protein binding is associated with long duration of action.

Clinical applications

1. Epidural administration for many surgical procedures and post-operative analgesia.
2. Central Blocks.
3. Peripheral nerve blocks like brachial plexus, sciatic nerve block, femoral nerve block and ankle block.
4. Labour epidural analgesia – Ropivacaine provides effective pain relief in labour. Due to its differential blockade ropivacaine is advantageous in labour analgesia.
5. Extradural anaesthesia for caesarean section.
6. Infiltration Anaesthesia.
7. Chronic pain management in cancer patients.

DEXMEDETOMIDINE HYDROCHLORIDE [25, 26, 31.]

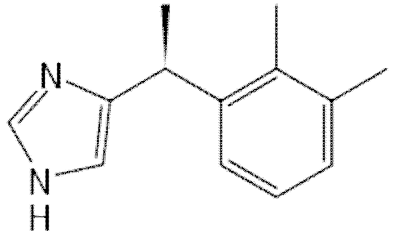
It is highly selective and more potent and α - 2 adrenoreceptor agonist with much greater affinity for α - 2 receptors over α-1(1620:1). It exerts analgesic, sedative, anxiolytic and hemodynamic stability. Its hemodynamic effects are due to sympatholytic action. This novel agent used as adjuvant during anaesthesia to reduce the hypnotic and opioid requirements for conscious sedation.

History

In the early 1960s the first α_2-adrenoceptor agonist was used for nasal decongestant purpose.

The introduction of clonidine as an antihypertensive drug in 1966.

In December 1999, dexmedetomidine was approved and used in humans for sedation, but only for a short period of time.



(S)-4-[1-(2, 3-Dimethylphenyl) ethyl]-3H-imidazole

Chemical: An imidazole derivative.

As a clear, colourless, isotonic solution containing dexmedetomidine base 100 µg/ml. It is preservative-free solution.

Physiochemical properties

Protein binding	: 94%
Metabolism	: Near complete hepatic metabolism to inactive metabolites
V_D	: 118 l/kg
Distribution half-life	: 6 minutes
Elimination half-life	: 2 hours
Excretion	: 95% of the metabolites excreted in the urine
Clearance	: 39 l/hr

Route of administration/doses

ICU sedation

The drug is administered by intravenous infusion commencing at 1 µg/kg for 10 minutes, then at 0.2 – 0.7 µg/ kg/hr. The duration should not exceed 24 hrs. It also been administered transdermally and intramuscularly.

Main action

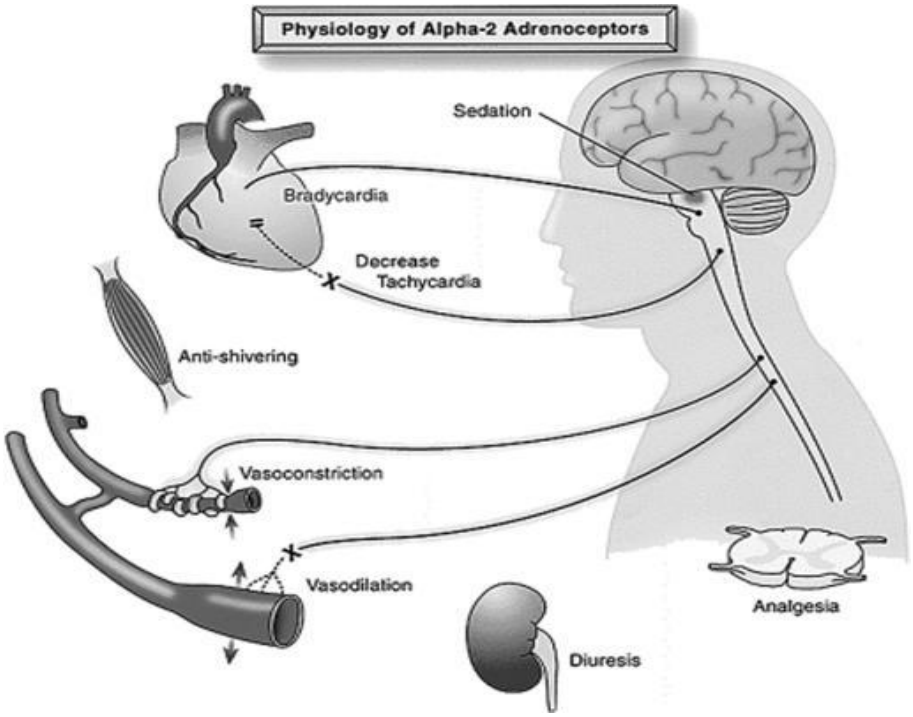
It has an analgesic, amnestic, sedative, anxiolytic properties.

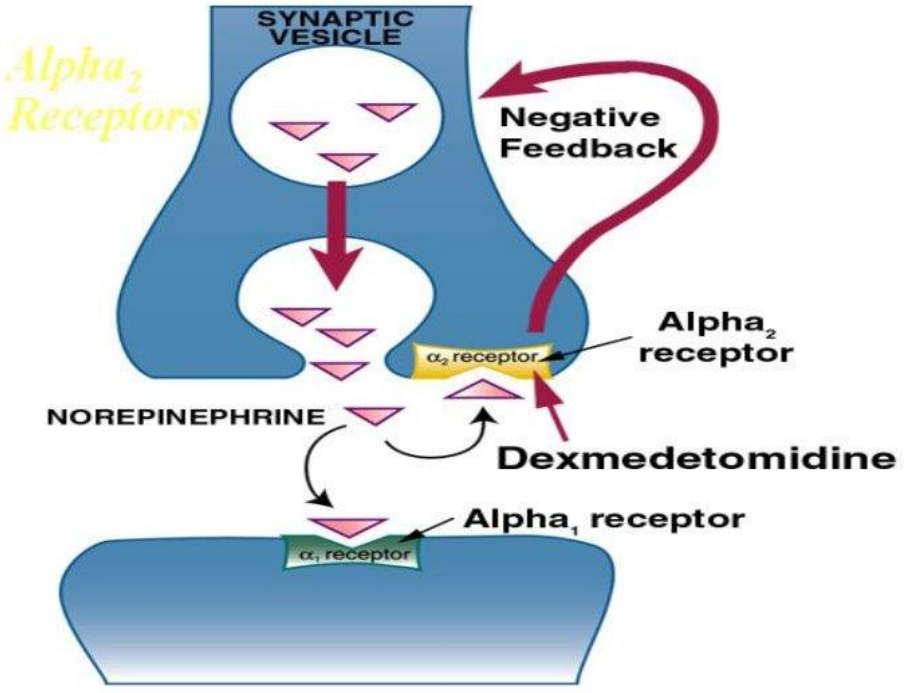
Mechanism of action

The main action of α–1 receptor is the regulation of vascular tone. It is widely distributed. The α-2 receptors are present in the peripheral as well as in the central nervous system which are pre and post synaptic receptors. Sedative actions are believed to be mediated primarily by post synaptic α-2 receptors. The main action of α-2 a receptors are sedative, anxiolytic and sympatholytic effects. They are mediated via G-protein inhibition of L-type calcium channels which are present in the post - synaptic receptors located in the locus coerelus. It appears to inhibit ion conductance through L-or P-type calcium channels and to facilitate conductance through voltage - gated calcium activated potassium channels. These effects are readily reversible by α -2 adrenergic antagonists like atipamezole.

Cognitive function does not alter with dexmedetomidine. When compared to propofol and thiopentone, they cause hyperpolarization of neurons through chloride channels opening.

MECHANISM OF ACTION





It produces analgesic action by an alpha–2 receptor which is present in the spinal cord and the locus coerelus. Substance P released from the modulation of spinal cord - dorsal horn which has more alpha – 2 receptors thereby producing the analgesic effect.

Decreased sympathetic output centrally by stimulation of α–2 adrenergic receptors produces more inhibitory neurons firing action.

In vascular system by Stimulation of α-2 receptors post synaptically produces vasoconstriction effect. Thus, dexmedetomidine has dual mode of action ie, central and peripheral. Centrally, it acts on the post synaptic α-2 inhibitory receptors, resulting in sympatholysis and sedation. The same action at the peripheral level leads to analgesic effect. In the peripheral nerves and autonomic ganglia it reduces the release of catecholamines leading to sympatholytic effect.

Decreased systemic vascular resistance and indirectly decreased myocardial contractility, cardiac output and systemic blood pressure. The initial hypertensive response is because of vasoconstriction effect mediated by peripherally located post synaptic alpha-2B action. This vasoconstriction effect can be prevented by slow administration of initial dose. Hypotension and bradycardia are caused by central pre-synaptic stimulation of alpha-2A with reduced norepinephrine release.

Pharmacokinetics

Following intravenous administration dexmedetomidine produces rapid distribution phase with distribution half-life about six minutes. The elimination half - life is approximately 2 hours and a clearance of 39L/h. Complete biotransformation involves both direct glucuronidation as well as cytochrome P450 (primarily by CYP2A6) mediated mechanism in the hepatic system. The metabolites do not have any clinical effects. They are excreted predominantly in the urine and little in the feces. Dose reduction may be needed in hepatic dysfunction. The excretion of the metabolite via the kidneys necessitates a dose reduction only when the Creatinine Clearance is less than 30ml per min, but half the dose reduction may be necessary with hepatic impairment. Even with continuous infusion more than 24 hours accumulation of drugs does not occur. Plasma concentration of therapeutic levels ranges between 0.3 to 0.6ng/ml.

Perioperative uses of dexmedetomidine

Premedication

It possesses anxiolytic, sedative, analgesic, antisialogogue and sympatholytic properties, which render it suitable as a premedication agent.

Intraoperative uses

- As adjunct to general anesthesia

The use of intraoperative dexmedetomidine may increase hemodynamic stability because of attenuation of the stress - induced sympathoadrenal responses to intubation, during surgery and during emergence from anesthesia.

- As adjunct to regional anesthesia

It shortened the onset time and prolonged the duration of the block and post-operative analgesia. The peripheral analgesic effects of dexmedetomidine that potentiate local anesthetics are mediated by α2A-AR binding to enhance the post-operative analgesia.

- In monitored anesthesia care (MAC)

It is a safe sedative alternative to benzodiazepine/opioid combinations in patients undergoing monitored anesthesia care for a multitude of procedures because of its analgesic, cooperative sedation, lack of respiratory depression properties.

- As a sole agent for procedural sedation.

A new role as a sole agent for procedural sedation is fast emerging mainly due to its faster onset of action, faster recovery and discharge times.

- Post-operative

Post operatively intravenous dexmedetomidine infusion is associated with a reduction in nausea and vomiting, reducing postoperative morbidity.

- For acute / chronic pain

The management of acute postoperative pain and chronic pain states, neuropathic pain, muscle spasticity and myofascial pain, sympathetically maintained pain such as complex regional pain syndrome (CRPS) and chronic headaches are due to its effects of opiate sparing action which has significant implications. It is evolving as an adjuvant analgesic, both as intravenous and intrathecal infusion, in cancer pain refractory to multiple treatment modalities.

Other uses

Prevention and treatment of emergence delirium, Alcohol withdrawal, shivering.

Precautions

Caution when used in patients with severe bradycardia, severe ventricular dysfunction (EF<30%), cardiac failure. Continuous blood pressure, electrocardiogram and oxygen saturation are recommended during infusion.

Toxicity

Hypotension, Hypertension, bradycardia, nausea, dry mouth.

ULTRASOUND GUIDANCE IN REGIONAL ANAESTHESIA[20, 21, 22.]

Supraclavicular plexus block was originally described by Kulenkampff in 1911. He introduced the concept of locating a nerve with paresthesia. Serious complication like pneumothorax due to blind technique because it mainly dependent on the anatomical landmarks. With advent of ultrasound supraclavicular block has become the most popular and common technique to provide anesthesia and analgesia to upper limb surgical procedures.

Non- invasive visualization of tissue structures by ultrasound technique, ensures the optimal spread of local anesthetic solutions around nerve structures. It hence improves the quality of analgesia and avoids the complication like inadvertent, intraneuronal and intravascular injection.

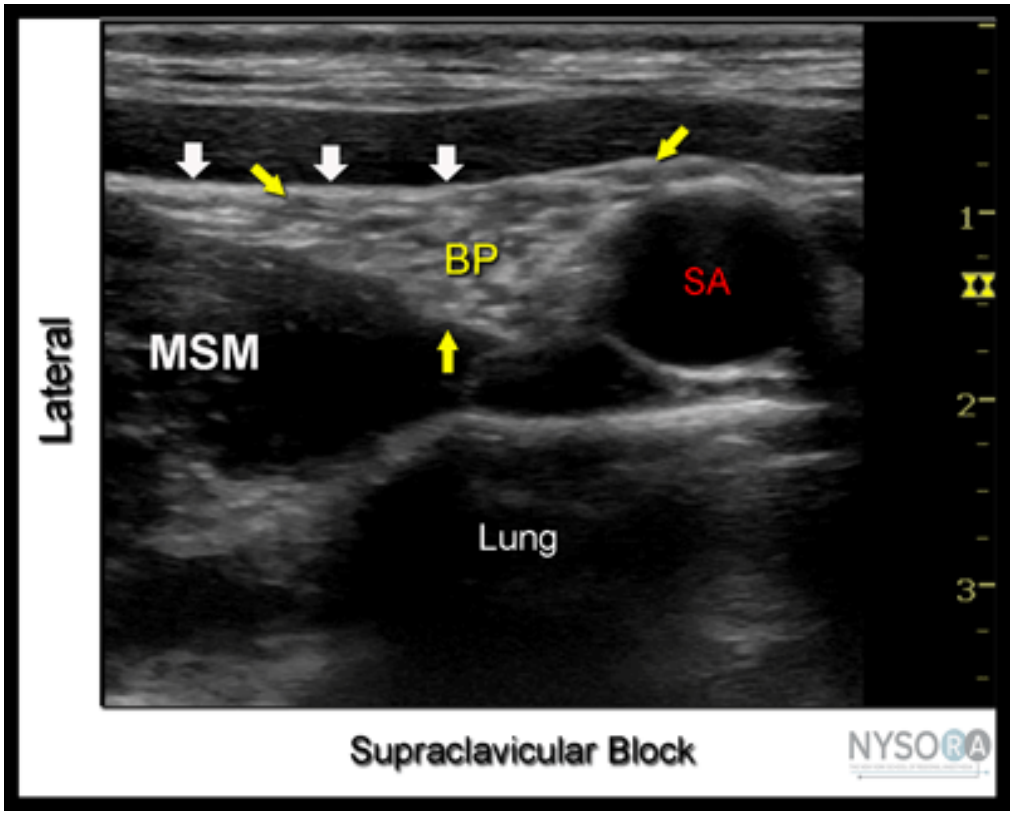
Principles

Ultrasound is a mechanical wave with frequencies over 20000 Hz sound generated and sent through tissues. Penetration into tissue is based in large part on the range of the frequency produced. Lower frequencies 2 MHz penetrate deeper than higher frequencies 10 MHz. As the sound passes through tissues it is either absorbed, reflected or allowed to pass through depending on the echodensity of the tissue. All ultrasound waves dissipate in tissues producing heat.

High frequency linear probe (8-15 MHZ)

Sonographic appearance of supraclavicular brachial plexus

(Bundle of hypo - echoic round nodules)



The listening part of the probe (a piezo electric crystal) like the generating part of the probe listens for reflections of the sound waves sent out and passes information to the processing unit.

The amount of energy reflected which is not absorbed or propagated equals density. Substances with a high water content like CSF and blood are very good conductors of sound. They reflect very poorly and are called echoluscent. Because they reflect very little of the sound, they appear as dark areas. Substances with low in water cotent like air, bone are poor sound conductors reflect all the sound energy and appear very bright. Substances which conduct sound between these two extremes appear darker to lighter depending on the amount of energy they reflect.

Ultrasound machine

The echoes received by the transducer are converted into visible dots by the ultrasound machine which helps in the formation of the anatomic image on the ultrasound screen. The ultrasound machines that are currently been used in the regional anesthesia provide a 2-D image. Recent advance of machine with 3- D image system are developed. It provides better visualization of local anesthetics solution distribution around the plexus and the identification of anatomical structures.

It consists of a transducer, control unit and display.

Transducers vary in size, shape, frequency range and number of piezoelectric crystals. For superficial block, high frequency transducer (7-15MHz) will provide better axial resolution. The presence of more piezoelectric crystal elements, produce the better the resolution. A lower frequency transducer (1- 5MHz) is appropriate for deeper blocks as there is less absorption and thus better signal from the deeper structure. Transducers with a small footprint (i.e. hockey stick transducers) are useful in children or where space is limiting. Wider with large footprint and curvilinear transducer allow for visualization of a bigger with large area. An aqueous gel is used as a coupling medium that is applied between the skin and the transducer for the elimination of the air layer.

Control unit allows for adjusting the depth of image, gain, focus to select different visualization modes etc. The ultrasound transducer incorporates a battery of piezoelectric crystals. When scanning the transducer switches quickly between transmitter and receiver mode. When in transmitting mode the piezoelectric crystals are stimulated by electrical energy vibrate and emit ultrasound waves. In the receiver mode the crystals are hit by the ultrasound waves reflected from the tissues. Finally, the mechanical stimulation of crystals is converted to electrical signals, which are processed and ultimately create the image we see on the screen.

Selecting the appropriate depth is the first step while scanning. The target structure should be in the center of the imaging field where the best resolution is possible. By manipulating the gain one can improve the image quality. The focus refers to the depth at which best lateral resolution is achieved.

Two needle insertion technique

The needle can be inserted in two ways. They are:

- In-plane technique
- Out-of-plane technique

In the first technique needle is to be kept in the plane of ultrasound beam, which results in the clear longitudinal view of the tip and needle shaft when it is advanced towards the target nerve.

In the next technique the needle must be inserted perpendicular to the transducer. The shaft of the needle is observed in a cross - section plane and a bright dot in the image can be seen. When compared to in-plane technique the needle tip is difficult to visualize and is not reliable in this method.

Sonographic appearance of peripheral nerves and various structures

The peripheral nerves appear hypo echoic (dark structures) or hyper echoic (bright structures) depends on

a) The size of the nerve

b) Sonographic frequency

c) Angle of ultrasound beam

The proximal parts i.e. nerve roots when scanning the interscalene area are rich in nerve tissue and appear black or hypo echoic. As the brachial plexus runs distally i.e. supraclavicular area, the proportion of connective tissue increases and the nerve has a grape like appearance. Further distally in the upper limb the connective tissue dominates and nerves appear more hyper echoic (honey comb appearance). Blood vessels can be identified by using Doppler colour ultra sound.

Veins	Compressible anechoic (black)
Arteries	Pulsatile enechoic (block)
Fat	Hypoechoic (black)
Fascia	Hyperechoic (black)
Muscle	Hypoechoic with hypereechoic striations (white and black)
Tendons	Hyperechoic (white)
Cartilage	Anechoic (black)
Nerves	Hyper or Hypoechoic
Local anesthetic	Anechoic (black)

REVIEW OF LITERATURE

1. M.S. Abrahams, M.F. Aziz et al (2009)[1] did a systematic review and meta-analysis of randomized controlled trials in ultrasound guided peripheral nerve block versus electrical neurostimulation method. They concluded that ultra sound improves the effectiveness of peripheral nerve blockade when compared to neurolocator for the identification of nerve.

2. Vincent W.S. Chan, Anahi Perlas et al(2007)[2] conducted a study in 188 patients using ultrasound guided(US), nerve stimulator(NS) and combined both (USNS). They proved that axillary brachial plexus block can be significantly improved by the guidance of ultrasound with or without concomitant use of nerve stimulation.

3. Vincent W.S. Chan, Anahi Perlas et al (2003)[3] 40 outpatients were assessed based on the ultrasound technology for supraclavicular brachial plexus blocks. Brachial plexus was identified using the ultrasound imaging technique. It also helps to guide the block needle to reach the target nerves and in the visualization of the pattern of local anaesthetic distribution. The block was successful after one attempt in 95% cases with one failure due to subcutaneous injection. There was no pneumothorax. They suggested that ultrasound probe with a high

resolution can accurately localise the brachial plexus and the structures near it in the supraclavicular region for peripheral nerve blockade.

4. Tomki Nishiyama, Higashi Omiya et al (2012)[4] compared the motor and sensory block by ropivacaine and bupivacaine in combination with lidocaine in 100 adult patients scheduled for repair of fracture of the upper extremity under interscalene block. They found that interscalene block combined with lidocaine, ropivacaine had slower onset of motor block and longer duration of both sensory and motor blocks than bupivacaine.

5. Esmaoglu A, Yegenoglu F, et al(2010)[5] added dexmedetomidine to levobupivacaine for axillary brachial plexus block and found that it shortens the time of onset in both sensorimotor block and prolonged the duration of block and duration of postoperative analgesia. Because it reduces the release of norepinephrine, leading to α-2 receptor independent inhibitory effects on nerve fibre action potentials.

6. Brummet CM, Hong EK, Janda AM, et al (2011)[7] showed that perineural dexmeditomidine added to ropivacaine for sciatic nerve block in rats prolonged the duration of analgesia by blocking the hyperpolarisation- activated cation current, which prevents the nerve from returning from a hyperpolarised state to resting membrane potential for subsequent firing.

7. Hickey, Hoffman J, Ramamurthy S,(1991) compared the effectiveness of 0.5% ropivacaine and 0.5% bupivacaine in 48 patients received a subclavian perivascular brachial plexus block for upper extremity surgery and concluded that both the drugs were equally effective in providing brachial anaesthesia.

8. Sadndya Agarwal, Ritu Agarwal et al (2014) compared the effects adding dexmedetomidine 100µg to 0.325% of bupivacaine (30 ml) for brachial plexus block by supraclavicular in 50 patients posted for upper limb surgeries. The study design was a prospective, randomized, double - blind, placebo-controlled trial. They found that dexmedetomidine when added to bupivacaine for supraclavicular block shortens the onset times for sensory and motor block and prolongs their duration. The added advantage of conscious sedation, hemodynamic stability and minimal side effects makes it a potential adjuvant for nerve blocks.

9. F.W. Abdulla and R. Brull (2013)[11], examined if perineural dexmedetomidine used as a local anaesthetic adjuvant for central neuraxial and peripheral nerve blocks can duration of analgesia be extended as compared to local anaesthesia alone. A total of 516 patients were analysed from nine randomised controlled trials. Five trials investigated dexmedetomidine as part of spinal anaesthesia and

four as part of a brachial plexus block. Sensory block duration was prolonged by 150 minutes with intrathecal dexmedetomidine. Perineural dexmedetomidine may prolong the mean duration of sensory block by 284 minutes. Motor block duration and time to first analgesic request were prolonged for both intrathecal and brachial plexus block. Dexmedetomidine produced reversible brachial plexus block in 7% of brachial plexus block patients, but no effect on the incidence of hypotension.

10. Marhofer D, Kettner SC et al (2013)[14], evaluated dexmedetomidine as an additive agent to ropivacaine in peripheral nerve blockade. Profound prolongation of ulnar nerve block was detected with perineural dexmeditomidine when added to 0.75% ropivacaine.

11. EL Saied et al (2000)[15], Conducted study in which axillary brachial plexus blockade was performed in 50 patients using 40ml ropivacaine 0.75%. Group (A) had 150µg clonidine and Group (B) 1ml normal saline added to the local anaesthetic. There was no difference in onset of sensory motor blockade. They concluded that the addition of 150µg of clonidine to ropivacaine for brachial plexus blockade prolongs motor and sensory block and analgesia without an increased incidence of side effects.

12. Klein SM, Greengrass RA et al. (1998), compared 30ml of 0.5% bupivacaine, 0.5% ropivacaine and 0.75% ropivacaine for interscalene brachial plexus block in 75 adult patients. Main purpose of this study was to determine the concentration and ideal long acting local anaesthetic drugs for interscalene brachial plexus block. The mean onset time of both motor and sensory blockade was <6 minutes in all groups. Duration of sensory block was similar in all groups. They concluded that there was no clinically important difference in times to onset and recovery of interscalene block for bupivacaine 0.5%, ropivacaine 0.5% and ropivacaine 0.75% when injected in equal volumes. Improvement in the time of onset or duration of analgesia of interscalene brachial plexus block cannot be achieved by increasing the concentration from 0.5% to 0.75% of ropivacaine.

MATERIALS AND METHODS

This study was carried out in the orthopedic surgery, Government Kilpauk Medical College and Hospital, Chennai after obtaining Institutional Ethical Committee approval.

Study design

This study designed as a prospective, randomized, double blind, comparative study. The local anesthetic solution prepared by an anesthesiologist who was not involved in administration of block and post-operative follow up. The anesthesiologist performing the block and observing the patient was blinded to the treatment group. The study may be unblinded at any point of time if any complications occurred for benefit of the patient.

Patient selection

The fifty adult patients both sexes age group of 20-60 years belonging to ASA I and II category and their weight ranging between 50-70 kgs of various types upper limb orthopedic elective surgeries under ultrasound guided brachial plexus block by supraclavicular approach who fulfill inclusion criteria were enrolled in this study.

Group selection

The patients were randomly assigned using "slips in the box technique" to either of the following groups.

Group R: 25 patients received 30 ml 0f 0.5% Ropivacaine+1ml saline.

Group RD: 25 patients received 30ml of 0.5% Ropivacaine+1ml (1/2ml of Dexmedetomidine with 1/2 ml of saline)

Inclusion criteria

- ASA I and II
- 20 – 60 years
- Both sexes
- Weight 50 – 70 kilograms
- Mid humerus, Elbow, Forearm and Hand surgeries

Exclusion criteria

- Patient refusal
- Coagulopathy
- ASA III and above
- H/O severe cardiovascular, pulmonary, kidney, liver disease
- neurological, psychiatric, neuromuscular disorders
- Infection / Sepsis / Allergy

- Pneumothorax
- Peripheral Neuropathy

Materials

- Sterile tray for regional block
- Sterile towel with gauze, 20 ml syringe, 10 ml syringe, 2ml syringe
- Drugs for the block 0.5 % Ropivacaine 30 ml, dexmedetomidine 50 mcg
- 22 G, 50mm short bevel insulated stimulating needle
- Ultra sound machine with high frequency (10-15 MHz) linear probe
- Equipment's and drugs for resuscitation.
- Equipment's and drugs for conversion to general anesthesia in the case of block failure.

Monitoring

- Pulse oximetry
- Noninvasive blood pressure
- Electrocardiogram

Methodology

Pre-operative preparation

All Patients were pre-operatively evaluated, clinically examined and proper investigations were done prior to assessment. Procedures were explained to the patient and informed consent was obtained. They were assessed with particular attention to any contraindications. Before the procedure VAS (Visual Analogue Scale) on 0-10cm was clearly explained to the patient for the assessment of post-operative pain. In this scale

i. 0 - indicate no pain

ii. 10 - indicate worst pain

Preoperative night patient was given with Tab. Alprazolam 0.5mg and Tab. Ranitidine 150 mg.

Conduct of anesthesia

On arrival of the patient in the operation room, preprocedure parameters blood pressure, heart rate and oxygen saturation were recorded and noted. In the opposite limb an intravenous access was obtained with 18G cannula and Ringer's lactate was started.

Positioning

The Patients was positioned supine with arm placed by the side. The head was turned facing 45º to the contralateral side to be blocked.

Scanning Technique, Nerve Localization and Needle Placement

The neck was cleaned with povidone iodine solution and draped with sterile towels. The anesthesiologist stands at the head end of the patient. Sterile gel was used between the probe and skin surface. In the coronal oblique plane the probe was kept in the supraclavicular fossa.

The pulsating hypo echoic subclavian artery was identified and confirmed by colour Doppler, lying above the hyper echoic first rib. While maintaining the view of the artery the probe was angled until both the first rib and the pleura were seen simultaneously to visualize these two structures.

Once the artery, rib, pleura and plexus were simultaneously in view the aim was to guide the needle inferior to the first rib, medial to the subclavian artery and superior to the nerves. In this area the lower trunk commonly lies. After local skin infiltration, the needle was entered by in - plane from lateral to medial until brachial plexus was reached. The supraclavicular brachial plexus was visualized as a group of hypo echoic nodules. The local anesthetic solution was injected after careful aspiration and spread was seen encircling the trunks. After injecting the local anaesthetic the block was tested for both

sensory (using pin prick) and motor (using muscle power) and was compared with the contralateral side.

Evaluation of the block

Sensory block was evaluated using 3-point scale by the pin prick method. After injecting the local anesthetic drug the sensory block was assessed at every minute in the dermatome areas corresponding to Median nerve, Radial nerve, ulnar nerve and Musculocutaneous nerve until the completion of sensory blockade.

Onset of sensory block means - Dull sensation to pin prick along the nerve distribution. (Grade-1)

Completion of sensory block means - Complete loss of sensation to pin prick. (Grade-2)

Grade	**Sensory block**
0	Sharp pin felt
1	Analgesia , dull sensation
2	Complete Anaesthesia, absence of sensation

Evaluation of motor block was done at every minute till complete motor blockade after the drug injection.

Evaluation of motor block done by

- Thumb abduction (Radial nerve)
- Thumb adduction (Ulnar nerve)
- Thumb opposition (Median nerve)
- Flexion of the elbow, supination and pronation of the forearm (Musculocutaneous)

Onset of motor blockade means Grade -1.

Peak motor block means Grade- 2.

Modified Bromage scale

This scale assessed the motor block for upper limb on a 3-point scale.

Grade	Motor blockade
0	Full flexion, extension of elbow, wrist and fingers, Normal.
1	Decreased motor power, can able to move the fingers only.
2	Inability to move the fingers, Complete block.

Incomplete block referred to - No analgesia in any of the segments supplied by Median, Radial, Ulnar and Musculocutaneous nerve after 30 minutes of drug injection.

Failed block - More than one nerve remained unaffected.

In this situation, general anesthesia was considered and not included for study.

Hemodynamic monitoring was done such as heart rate, blood pressure and oxygen saturation continuously throughout the surgery and every 60 minutes post - operative period.

Sedation of patient was assessed by the **Ramsay Sedation Scale**.

Score	**Definition**
1	Patient anxious and agitated or restless or both
2	Patient cooperative, oriented and tranquil
3	Patient responds to commands only
4	Patient has a brisk response to a light glabellar tap or loud auditory stimulus
5	Patient asleep, sluggish response to light glabellar tap or loud auditory stimulus
6	Patient doesn't respond to painful stimulus

Assessment of blood loss was done and fluid was administered as per the loss. Duration of surgery was noted.

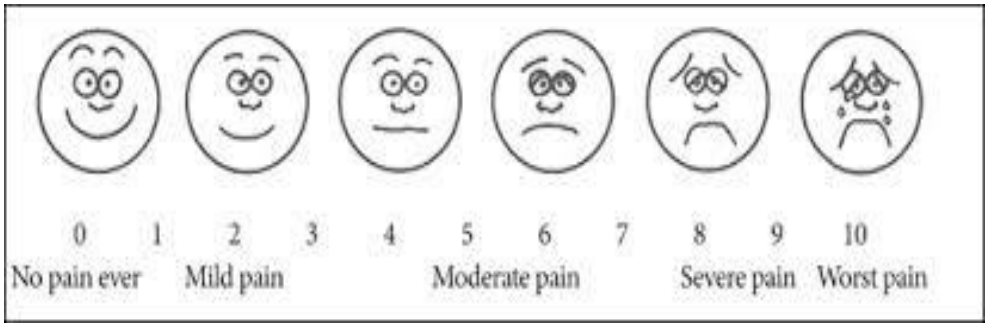
Patients were assessed for duration of analgesia as per Visual Analogue Scale. After the surgery it was monitored every 1 hour until the score reaches - 5. The rescue analgesia was given with parental use of Diclofenac injection when the VAS reaches 5 and the time of the injection was recorded.

All patients were observed for any side-effects like nausea, vomiting, dryness of mouth and complications like pneumothorax, hematoma, local anesthetic toxicity and post - block neuropathy in the intra - and post-operative periods. The patient was requested to note the subjective resolution of sensation and movements. Finally it was counter checked by the anesthesiologists.

Time interval between the completion of local anaesthetic solution administration and the complete resolution of anaesthesia on all nerves was considered as the duration of sensory block (grade0).

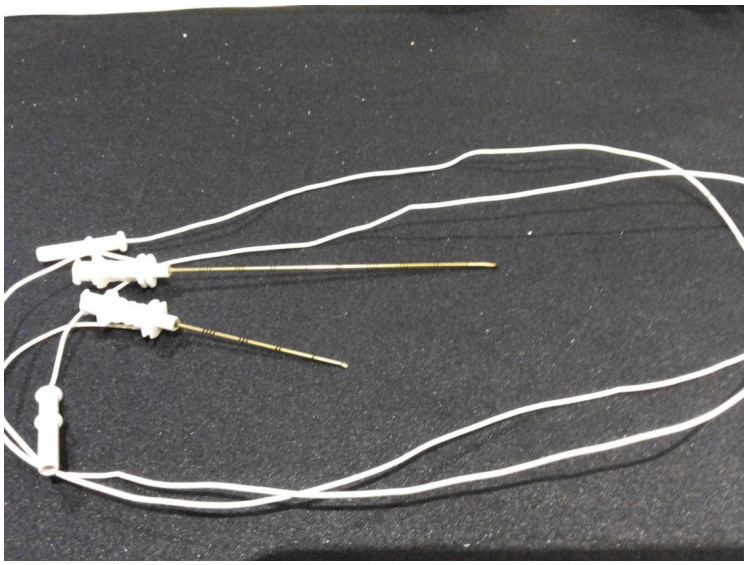
The time interval between the completion of local anaesthetic administration and the recovery of complete motor function of the hand and forearm was considered as the duration of motor block (grade 0).

VISUAL ANALOG SCALE



Ultrasound machine with linear transducer – Imaging the supraclavicular brachial plexus by an in-plane technique

22 Gauge Short bevel insulated stimulating needle



Drugs

Statistical Analysis

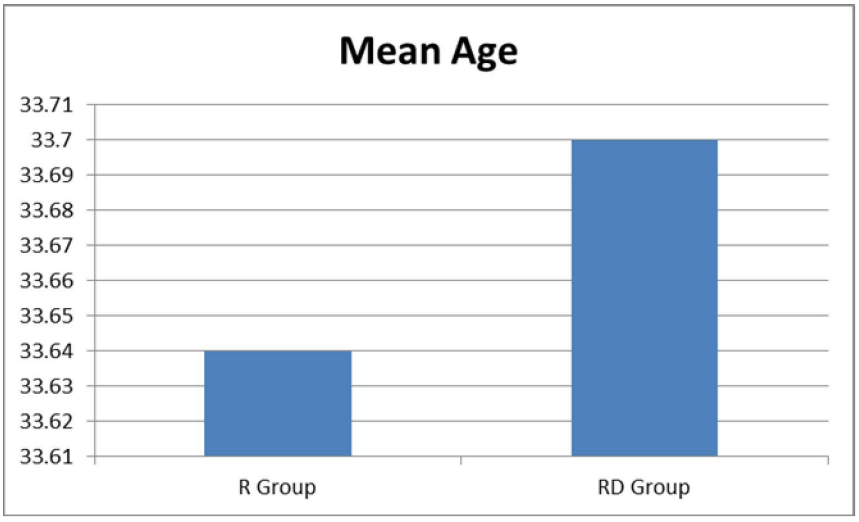
All the data were subjected to statistical significant estimation.

The parameters of age, weight, total time taken for surgery, heart rate, blood pressure, oxygen saturation, time taken for sensory and motor blockade, offset time for sensory and motor blockade and total time of analgesia were analyzed by Independent t-test. Chi-square test was used for sex and ASA. A P-value less than 0.05 were considered statistically significant.

OBSERVATION AND RESULTS

There was no statistically significant difference between the demographic data (age, Sex, weight) and ASA grade, duration of surgery.

Parameters	R Group	RD Group	T value	P value
Age (Years)				
Mean	33.64	33.70	-0.033	0.974
SE	1.68	1.73		
Weight (kg)				
Mean	69.16	69.16	0.00	1.00
SE	4.35	4.35		
Gender				
Male	16	18	0.37	0.544
Female	9	7		



The mean age of the subjects in R group was 33.64 years and it was 33.7 years in RD group.

P value- 0.794 : Not statistically significant.

ASA Physical Status

Parameters	R Group	RD Group	Chi square	P value
ASA				
I	22	21	0.17	0.683
II	3	4		

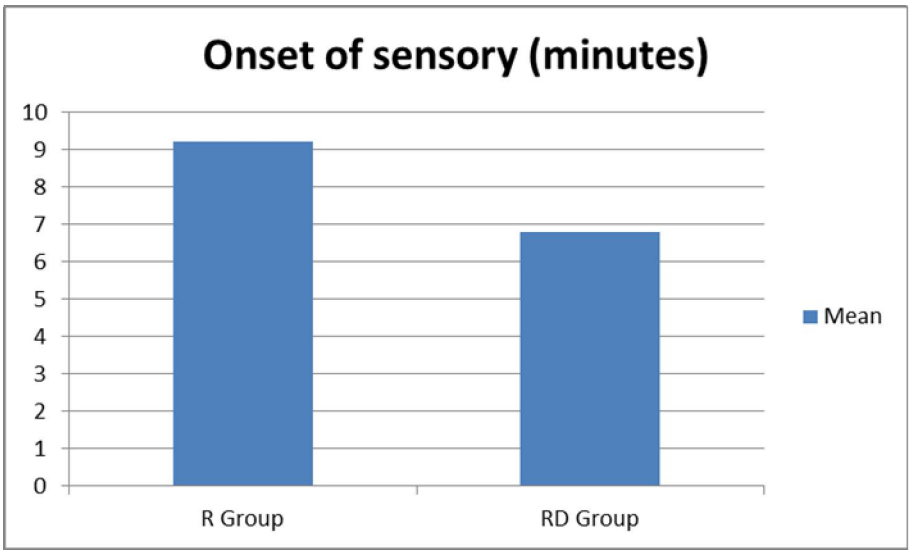
DURATION OF SURGERY

Duration of surgery (min)	R Group	RD Group	T value	P value
Mean	162.0	162.0	0.00	1.00
SE	3.74	3.21		

There were no statistically significant values observed in ASA physical status and duration of surgery in both the groups.

ONSET OF SENSORY BLOCK

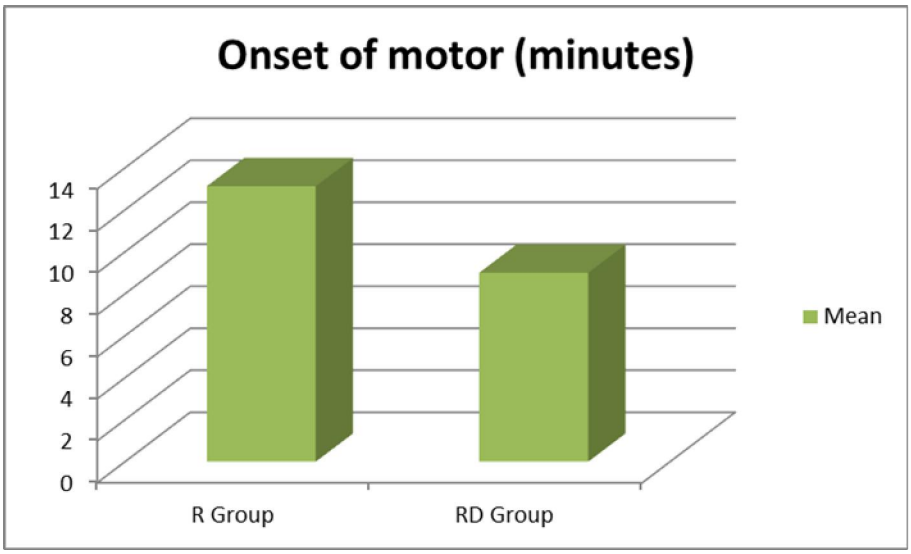
Onset of sensory (minutes)	R Group	RD Group	T value	P value
Mean	9.2	6.8	12.0	0.001
SE	0.15	0.13		



The mean onset of sensory block was 9.2 minutes in R group compared to 6.8 minutes in the RD group. This difference in means was found to be statistically significant.

ONSET OF MOTOR BLOCK

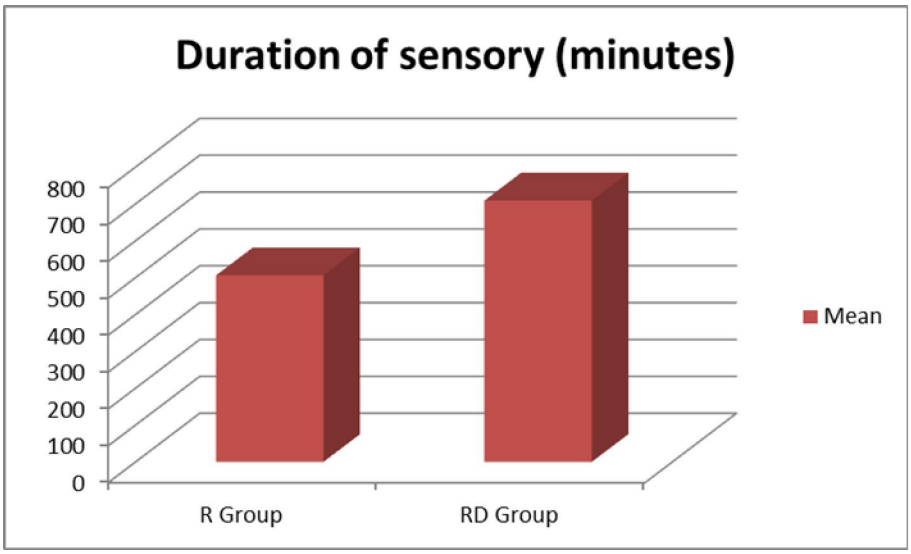
Onset of motor (min)	R Group	RD Group	T value	P value
Mean	13.12	9.0	18.232	0.001
SE	0.18	0.14		



The mean onset time of motor block was 13.12 minutes in R group compared to 9.0 minutes in the RD group. This difference in means was found to be statistically significant.

DURATION OF SENSORY BLOCK

Duration of sensory (minutes)	R Group	RD Group	T value	P value
Mean	506.2	709.0	-26.547	0.001
SE	3.71	6.68		

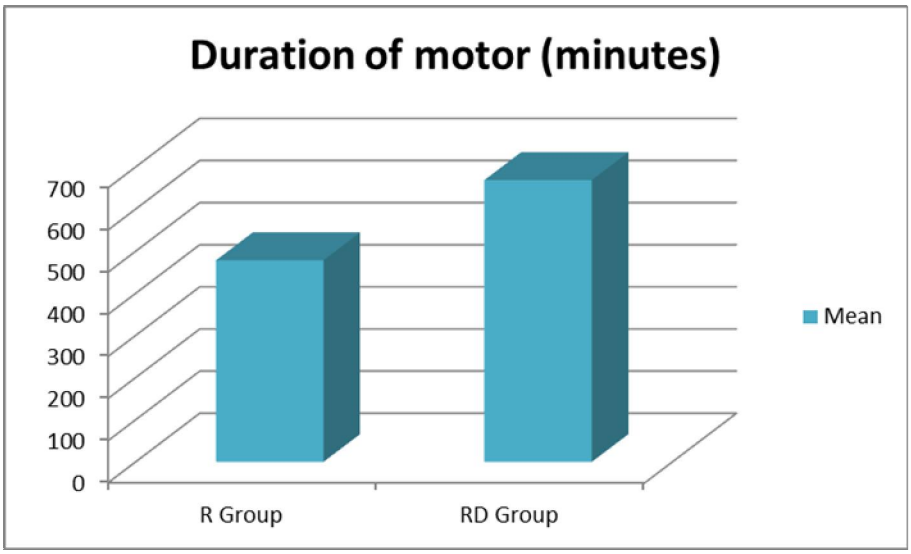


The sensory block duration (mean) of the subjects in group R was 506.2 minutes and it was 709 minutes in RD group.

P value - 0.001 : statistically significant.

DURATION OF MOTOR BLOCK

Duration of motor (min)	R Group	RD Group	T value	P value
Mean	478.8	669.2	-34.199	0.001
SE	3.74	4.12		

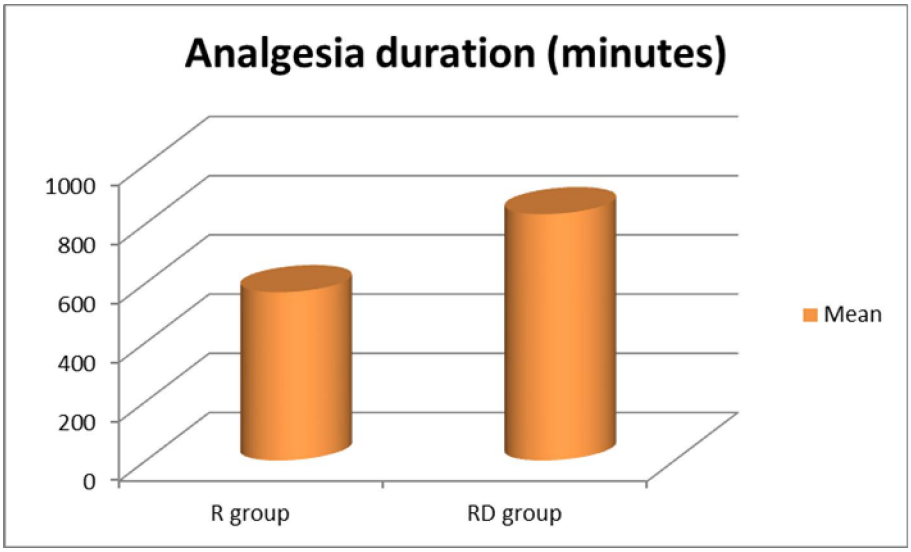


The motor duration (mean) of block of the subjects in group R was 478.8 minutes and it was 669.2 minutes in RD group.

P value -0.001 : statistically significant.

DURATION OF ANALGESIA

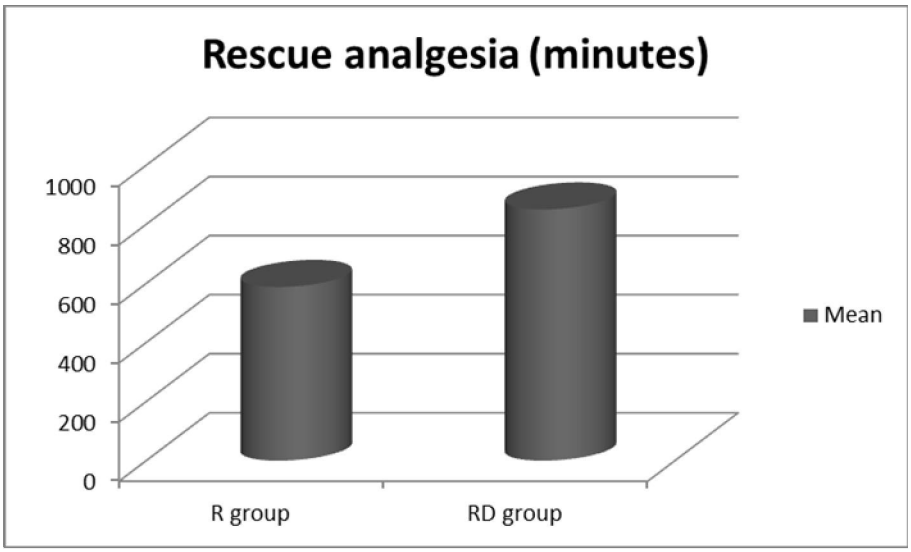
Analgesia duration (minutes)	R Group	RD Group	T value	P value
Mean	568.20	831.80	-34.998	0.001
SE	3.15	6.84		



The analgesia duration (mean) of group R was 568.2 minutes whereas it was 831.8 minutes among subjects of RD group. This difference is found to be statistically significant.

RESCUE ANALGESIA

Rescue analgesia (minutes)	R Group	RD Group	T value	P value
Mean	588.00	850.40	-34.682	0.001
SE	2.92	6.98		

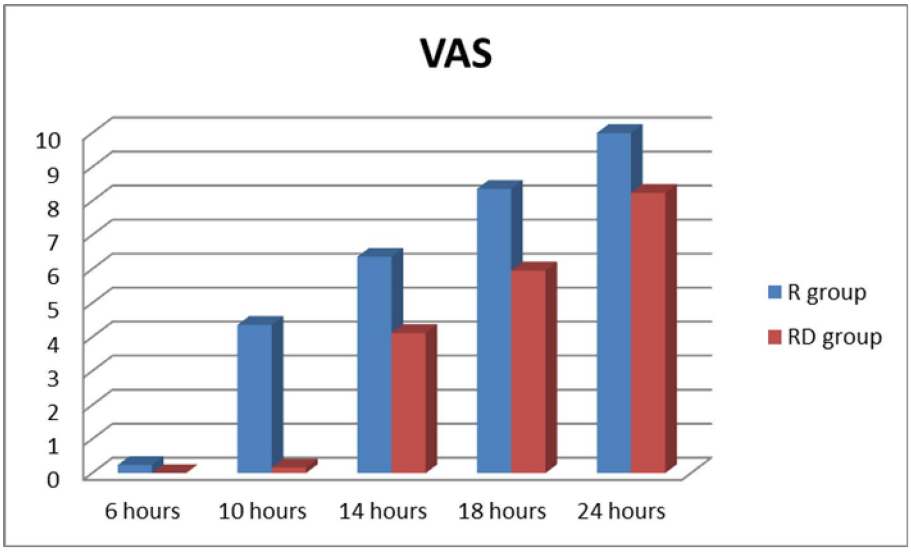


The mean time of rescue analgesia was found to be 588 minutes among subjects in group R, while the mean time of rescue analgesia was 850.4 minutes among RD group subjects.

P value-0.001 : statistically significant.

VISUAL ANALOG SCALE

Parameters	R Group	RD Group	T value	P value
VAS 6 hours				
Mean	0.24	0	2.753	0.011
SE	0.09	0		
VAS 10 hours				
Mean	4.36	0.16	30.851	0.001
SE	0.1	0.09		
VAS 14 hours				
Mean	6.36	4.12	18.931	0.001
SE	0.1	0.07		
VAS 18 hours				
Mean	8.36	5.96	17.955	0.001
SE	0.1	0.09		
VAS 24 hours				
Mean	10	8.24	13.266	0.001
SE	0	0.13		



The mean VAS of the R group when compared to RD group at 6, 10, 14, 18 and 24 hours were found to be statistically significant.

OBSERVATION OF HAEMODYNAMIC VARIABLES

Parameters	R Group	RD Group	T value	P value
HR 0 min				
Mean	92.96	93.4	-0.877	0.385
SE	0.39	0.32		
SBP 0 min				
Mean	131.52	131.52	0	1
SE	0.49	0.49		
DBP 0 min				
Mean	88.8	88.64	0.142	0.887
SE	0.79	0.8		
SpO2 0 min				
Mean	99.8	99.84	-0.361	0.72
SE	0.08	0.07		

Parameters	R Group	RD Group	T value	P value
HR 15 min				
Mean	85.28	76.56	10.855	0.001
SE	0.38	0.71		
SBP 15 min				
Mean	130	125.36	7.25	0.001
SE	0.5	0.4		
DBP 15 min				
Mean	84.64	74.72	14.399	0.001
SE	0.47	0.5		
SpO2 15 min				
Mean	99.76	99.8	-0.335	0.739
SE	0.09	0.08		

Parameters	R Group	RD Group	T value	P value
HR 30 min				
Mean	83.84	70	23.101	0.001
SE	0.32	0.5		
SBP 30 min				
Mean	129.04	107.84	31.016	0.001
SE	0.61	0.3		
DBP 30 min				
Mean	83.84	68.48	32.074	0.001
SE	0.42	0.24		
SpO2 30 min				
Mean	99.76	99.84	-0.696	0.49
SE	0.09	0.07		

Parameters	R Group	RD Group	T value	P value
HR 60 min				
Mean	83.6	66.48	30.089	0.001
SE	0.38	0.42		
SBP 60 min				
Mean	128.96	105.84	43.651	0.001
SE	0.46	0.26		
DBP 60 min				
Mean	83.44	66.64	47.863	0.001
SE	0.29	0.19		
SpO2 60 min				
Mean	99.76	99.84	-0.696	0.49
SE	0.09	0.07		

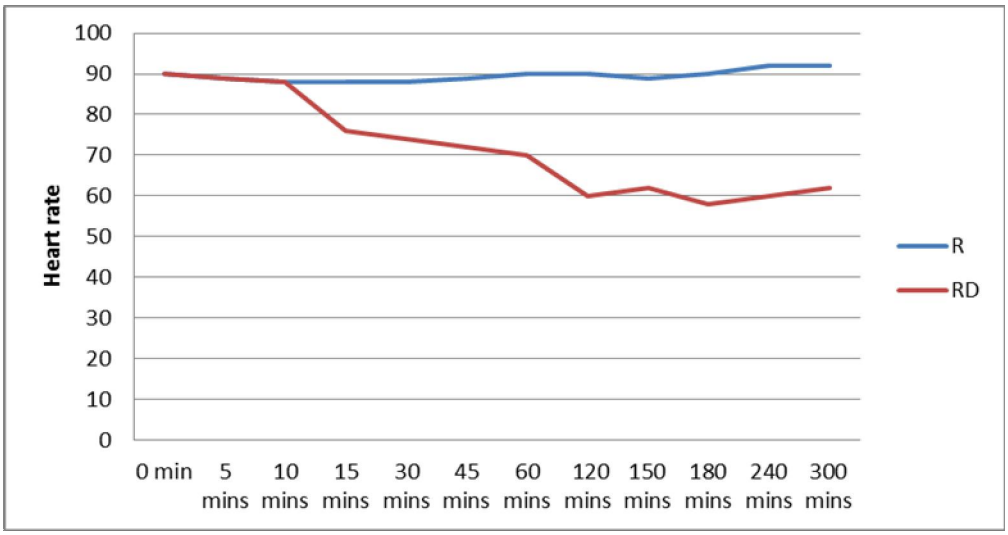
Parameters	R Group	RD Group	T value	P value
HR 120 min				
Mean	83.28	63.92	29.94	0.001
SE	0.36	0.54		
SBP 120 min				
Mean	128.32	104.96	39.828	0.001
SE	0.53	0.26		
DBP 120 min				
Mean	82.16	65.92	45.873	0.001
SE	0.23	0.27		
SpO2 120 min				
Mean	99.84	99.88	-0.4	0.691
SE	0.07	0.07		

Parameters	R Group	RD Group	T value	P value
HR 180 min				
Mean	82.48	62.72	39.095	0.001
SE	0.39	0.32		
SBP 180 min				
Mean	127.92	106	41.948	0.001
SE	0.47	0.23		
DBP 180 min				
Mean	82	65.2	31.749	0.001
SE	0.28	0.45		
SpO2 180 min				
Mean	99.8	99.92	-0.463	0.646
SE	0.07	0.06		

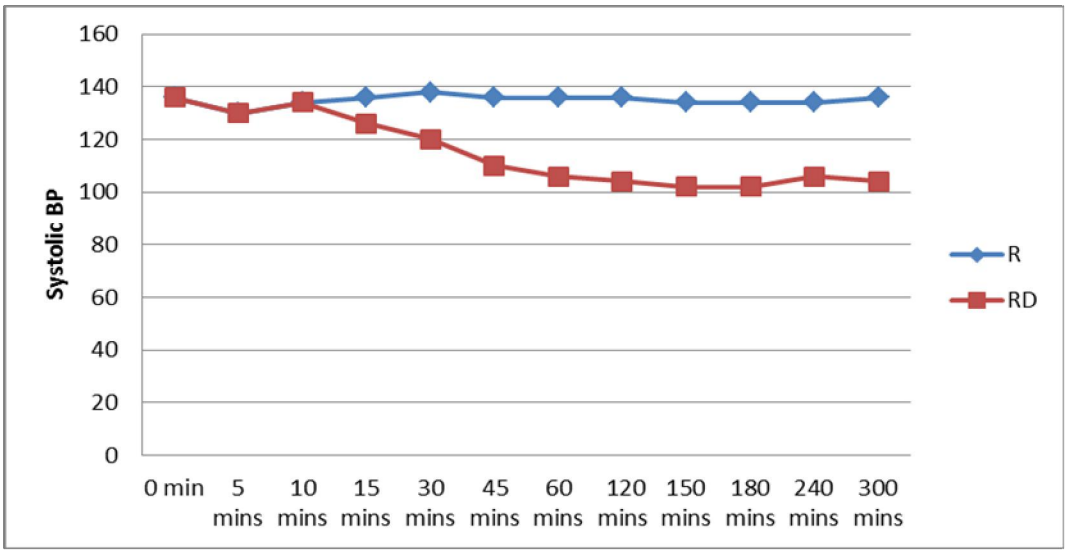
Parameters	R Group	RD Group	T value	P value
HR 240 min				
Mean	82.32	62.16	30.578	0.001
SE	0.47	0.46		
SBP 240 min				
Mean	127.6	106.96	36.396	0.001
SE	0.53	0.2		
DBP 240 min				
Mean	81.76	67.2	43.715	0.001
SE	0.27	0.2		
SpO2 240 min				
Mean	99.8	99.92	-1.216	0.231
SE	0.08	0.06		

Parameters	R Group	RD Group	T value	P value
HR 300 min				
Mean	81.84	64.48	43.043	0.001
SE	0.32	0.24		
SBP 300 min				
Mean	127.6	108.24	41.503	0.001
SE	0.48	0.13		
DBP 300 min				
Mean	81.68	67.6	39.192	0.001
SE	0.28	0.23		
SpO2 300 min				
Mean	99.8	99.92	-1.216	0.231
SE	0.08	0.06		

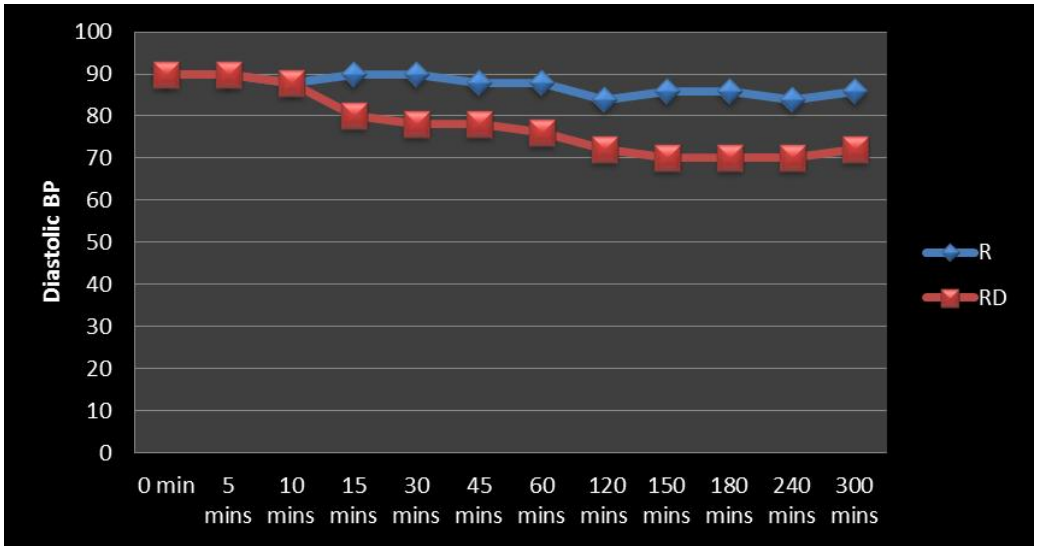
Trend line of Heart rate in both the groups



Trend line of systolic BP among the study subjects

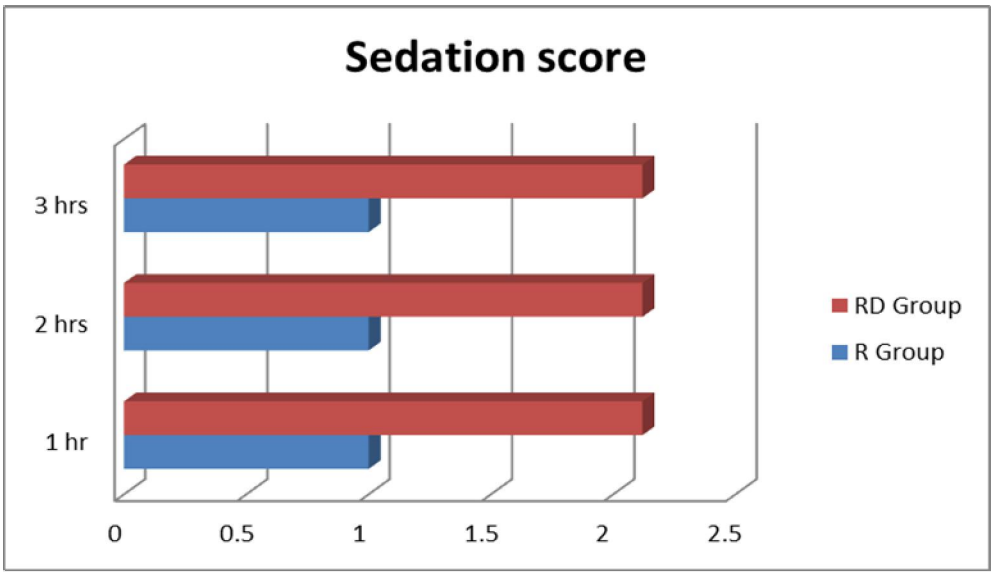


Trend line of diastolic BP among the study subjects



SEDATION SCORES

Parameters	R Group	RD Group	T value	P value
Sedation score 1 hour				
Mean	1.00	2.12	-16.885	0.001
SE	0.00	0.07		
Sedation score 2 hours				
Mean	1.00	2.12	-16.885	0.001
SE	0.00	0.07		
Sedation score 3 hours				
Mean	1.00	2.12	-16.885	0.001
SE	0.00	0.07		



The sedation score (mean) among the subjects in R group was 1 and the mean sedation score in group RD was 2.12 respectively. The difference was found to be statistically significant.

DISCUSSION

This prospective randomized double blind study was done in patients undergoing elective upper limb orthopaedic surgery under supraclavicular brachial plexus block.

Brachial plexus blockade provide an excellent alternative technique to general anaesthesia for upper limb surgical procedures. It not only offers excellent intraoperative pain relief but also good post-operative analgesia. Supraclavilar technique was chosen for this study because it provides a rapid onset, dense and predictable anaesthesia with high success rate.

Technique

In this study we used ultra sound guided supraclavicular blocks. This technique allows the direct visualisation of nerves and anatomical structures. Avoidance of complication and reduce the dose of local anaesthetic agents.

M.S. Abrahams et al[1] concluded that ultra sound improves the quality of blockade when compared to methods that use the peripheral nerve stimulator for nerve identification.

Vincent W.S.Chan et al[2] conducted a study in 188 patients and demonstrated that axillary brachial plexus block significantly improved the success rate under the guidance of ultrasound with or without nerve stimulation.

Hickey et al[7] studied subclavian perivascular brachial plexus block using 30 ml of 0.5% ropivacaine under elicitation of paraesthesia technique for localisation of nerve. They found that the peak onset of sensory blockade was 28 minutes. In this study the peak onset of sensory blockade was 9.2 minutes. The difference may be due to difference in technique used for the localisation of brachial plexus. They used elicitation of paraesthesia for localisation of nerve plexus, which was not as accurate as use of ultra sound in our study.

Drugs

Ropivacaine is a long - acting regional anaesthetic that is structurally related to bupivacaine. It is a pure S (-) enantiomer, unlike bupivacaine. It developed for the purpose of reducing potential toxicity and improving relative sensory and motor block profiles. Ropivacaine has lower lipid solubility and have produced less central nervous system and cardiac toxicity than bupivacaine for which it is gaining popularity over bupivacaine for peripheral neural blockade when large volumes of local anesthetic are required. Ropivacaine is also used in the chronic pain management.

Ropivacaine is as effective as bupivacaine and levobupivacaine when used in peripheral nerve blocks. But it is less potent than bupivacaine when used in epidural or Intrathecal route. Clinically adequate doses of ropivacaine appear to be associated with a lower grade of motor block than bupivacaine.

Ropivacaine is considered as an important option for regional anaesthesia, postoperative pain management and labour analgesia due to the following reasons:

- Efficacy
- Lower propensity for motor block.
- Reduced potential for central nervous system toxicity and cardio toxicity.

In our study, the mean onset time of sensory and motor was quicker in group RD. The mean duration of sensory (709 minutes), motor (669.2 minutes) in group RD, whereas in group R the mean duration of sensory (506 minutes), motor (478.8 minutes). The duration of analgesia was extended in Ropivacaine Dexmedetomidine group than in Ropivacaine group.

El Saied et al[15] concluded that the adding additives like clonidine (150 µg) to ropivacaine for brachial plexus blockade extends the duration of sensorimotor and that of analgesia without any side effects.

Klein et al[18] compared the efficacy of bupivacaine 0.5% and different concentration of ropivacaine 0.5%, ropivacaine 0.75%. They used 30ml in each group. They explored that there wasn't any significant difference in time of onset and recovery. On increasing the concentration from 0.5% to 0.75% of ropivacaine, it did not show any improvement in the onset or duration of

analgesia. In this study, we used 0.5% of ropivacaine 30ml which was similar to Klein et al study.

Post-operative analgesia can be extended by continuous catheter based technique. But it needs extra time, skill, cost and catheter related complications.

Pharmacologically active d-isomer of medetomidine is known as Dexmedetomidine[16].It is more selective towards α-2 adrenoceptor agonist with α-2:α-1 binding selectivity ratio of 1620:1 and decreasing the unwanted side effect of α-1 receptors. High selectivity for α-2 receptors mediates analgesia, sedation and anxiolysis.

Memis[12] and colleagues first proposed dexmeditomidine, α-2 adrenoreceptor agonist when used as an additive agent to local anaesthetics with an ability of prolonging the sensory and motor block duration.

Esmaoglu[5] et al. added dexmedetomidine to levobupivacaine for axillary brachial plexus block and showed that it shortens the time taken for onset of sensorimotor block and prolongs the time of blockade and that of post-operative analgesia.

A study by **Obayah**[13] and colleagues during greater palatine nerve block for cleft palate surgery added an adjuvant dexmedetomidine to bupivacaine solution. When bupivacaine mixed with dexmedetomidine

provided lower pain scores and prolonged analgesia with no negative effects on hemodynamics when compared to bupivacaine alone.

Several animal studies have investigated the analgesic effects of dexmeditomidine as an adjuvant. A study by **Brumett**[8] et al. showed that when dexmeditomidine used with bupivacaine, increases the duration of bupivacaine anaesthesia and analgesia of sciatic nerve block in rats without any damage to the nerve.

The above studies show that selective α_2 – adrenoceptor agonist like clonidine or dexmedetomidine when added as adjuvant to ropivacaine in different peripheral nerve block potentiates the sensorimotor blockade.

The mechanism of action is probably due to

- The peripheral action of α_2 agonists produce analgesia by decreasing the norepinephrine release, leading to α_2 - receptor independent inhibitory effects on nerve fibre action potentials.
- The central action of α2 agonists produces sedation and analgesia by inhibition of substance P release in the nociceptive pathway.
- Also by activation of α_2 – adrenoceptors in locus coeruleus.

Therefore, the dexmedetomidine action via a simillar mechanism of action when added with the local anesthetic solutions leading to a long lasting action.

Adding adjuvant like dexmeditomidine to ropivacaine prolonging the analgesia with one shot block can result in a longer time of post - operative analgesia and can avoid continuous catheterization.

The added advantage of conscious sedation, hemodynamic stability and the lack of significant side effects like respiratory depression make Dexmedetomidine better choice as an adjuvant for supraclavicular brachial plexus block.

None of the patients in group RD required sedation intra - operatively. They were comfortable throughout the surgery with arousable sedative effect. This is due to partial vascular uptake of the drug and its transport to the central nervous system. As α2 agonists produce sedation by central action and in the nociceptic pathway at the level of dorsal root neuron causes substance P release inhibition and in the locus coerelus α2 adrenoceptor activation. The limited duration of sedation could be explained by the fact that it is highly lipophilic and diffuses faster in to the blood vessels by rapid clearance and short half-life. In our study, the highest sedation score was 3. No patient required airway assistance due to sedation.

Haemodynamic variables like heart rate, blood pressure, oxygen saturation was significantly low in group RD from the 15^{th} minutes of the onset of blockade. They were stable throughout the surgery.

Only two patients had heart rate <60/min. But no patient required Inj.Atropine or vasopressor support. The hypotension was only mild and corrected with intravenous crystalloids.

No complications were observed.

SUMMARY

On comparing Ropivacaine 0.5% and Ropivacaine 0.5% with dexmedetomidine 50µg in Ultrasound Guided Supraclavicular Brachial plexus Block for Upper limb Orthopedic Surgery the following findings were observed.

1. Onset of sensory and motor blockade was quicker in group RD (Ropivacaine with Dexmedetomidine) when compared to group R (Ropivacaine).
2. The mean duration of the sensory and motor blockade was significantly prolonged in group RD (Ropivacaine with Dexmedetomidine).
3. The mean duration of analgesia was significantly prolonged in group RD (Ropivacaine with Dexmedetomidine).
4. Rescue analgesia was needed earlier in group R (Ropivacaine) than that of group RD (Ropivacaine with Dexmedetomidine).
5. Hemodynamic parameters were significantly low from 15^{th} minutes after the onset of blockade in group RD (Ropivacaine with Dexmedetomidine) than in group R (Ropivacaine).
6. Sedation was statistically significant in group RD (Ropivacaine with Dexmedetomidine) in the intraoperative period.
7. There was no complication seen in both the groups during the study.

CONCLUSION

In conclusion, Dexmedetomidine 50µg when added as an adjuvant to Ropivacaine 0.5% for supraclavicular brachial plexus block hastens the onset of sensory and motor blockade and prolongs the duration of sensorimotor blockade and provides a longer pain - free period when compared to Ropivacaine alone.

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PROFORMA

Name: Age/Sex: Wt: ASA Status:

IP No: Surgery: Duration of Surgery:

Group R/RD:

Preoperative examination

PR: BP: SPO2: CVS: RS: Airway:

Investigation: Hb: BT: CT:

Onset of block Sensory: Motor:

Completion of block Sensory: Motor:

Duration of Analgesia

		0 min	5 min	10 min	15 Min	20 min	30 min	1 hr	2 hr	3 hr	4 hr	5 hr	6 hr	12 hr	24 hr
Vital	**PR**														
	BP														
	RR														
	SpO2														
Sens&Motor Score															
Radial Nerve	**S**														
	M														
Median Nerve	**S**														
	M														
Ulnar Nerve	**S**														
	M														
Musculocut. Nerve	**S**														
	M														

Sedation Score	**Baseline**	**30 mins**	**60 mins**	**90 mins**	**120 min**	**180 min**	**240 min**

Time of Rescue analgesia/ Post op.	**1 hr**	**2 hrs**	**3 hrs**	**4 hrs**	**5 hrs**	**6 hrs**

Complication:

GROUP - R (Ropivacaine)

S. No		0 min					15 mins				30 mins				60 min				120 mins				180 mins				240 min				300 mins			
	HR	SBP	DBP	SPO2	HR	SBP	DBP	SPO2	HR	SBP	DBP	SPO2	HR	SBP	DBP	SPO2	HR	SBP	DBP	SPO2	HR	SBP	DBP	SPO2	HR	SBP	DBP	SPO2	HR	SBP	DBP	SPO2
1	90	136	90	100	88	136	90	100	88	138	90	99	90	136	88	99	90	136	84	100	90	134	86	100	92	134	84	100	86	132	82	99
2	92	134	90	100	86	134	88	100	82	136	88	99	88	134	86	100	86	134	84	100	84	134	84	100	86	134	84	99	86	132	82	100
3	90	132	92	99	88	130	86	99	84	134	86	100	82	132	84	100	84	132	84	100	84	132	84	100	84	134	84	100	84	132	82	100
4	91	130	88	100	86	128	84	100	82	128	84	100	82	130	82	100	82	130	82	100	82	130	82	100	82	130	84	100	82	132	82	100
5	93	130	90	100	86	128	82	99	84	128	82	100	82	130	84	100	84	130	84	99	82	128	82	100	82	126	82	100	82	128	82	100
6	91	128	92	100	84	126	84	99	86	126	84	100	84	128	84	99	84	126	82	100	82	128	82	100	82	128	82	99	82	126	84	99
7	92	130	94	100	84	130	86	100	82	128	84	100	82	130	84	99	82	128	82	100	82	126	82	100	82	128	82	100	80	126	84	100
8	96	134	90	100	86	132	88	100	84	130	86	100	84	128	84	100	82	126	82	100	80	126	82	100	80	126	82	100	80	126	84	100
9	94	132	92	100	86	130	82	100	84	128	82	99	84	128	82	100	82	126	82	100	82	126	82	100	82	128	80	99	80	126	84	99
10	93	136	86	99	84	132	82	100	84	130	82	100	82	130	82	100	82	128	80	100	82	126	84	100	80	126	82	100	82	128	82	100
11	94	128	84	100	86	126	82	100	84	126	84	100	84	128	84	100	84	126	82	100	82	128	82	99	82	126	82	100	82	128	80	100
12	91	130	80	100	88	128	84	99	86	130	84	99	84	128	84	100	84	128	82	100	84	126	80	99	82	126	80	100	82	128	80	100
13	95	134	82	100	84	132	86	100	82	126	84	100	82	128	82	99	82	128	82	100	80	126	80	100	82	126	82	100	82	126	80	100
14	94	128	80	100	86	126	86	100	84	126	82	100	84	130	84	100	82	128	82	100	82	128	82	100	80	128	82	100	82	126	82	100
15	93	130	86	100	84	128	84	100	86	128	82	100	84	130	84	99	82	130	82	100	82	126	82	100	82	126	82	100	82	126	82	100
16	90	132	90	100	84	130	86	100	82	128	84	100	82	128	82	100	84	130	84	100	82	128	82	100	82	126	82	100	82	126	82	100
17	91	134	92	100	88	132	82	99	84	130	82	100	84	128	82	100	82	128	82	100	84	128	82	100	82	126	82	100	80	126	82	100
18	92	130	90	99	86	130	82	100	84	128	82	99	84	126	84	100	84	126	80	100	82	126	82	100	82	126	80	100	80	130	80	100
19	94	132	92	100	84	132	86	100	82	130	84	100	82	128	82	100	82	126	82	100	82	128	80	100	82	128	80	99	80	128	80	100
20	95	130	86	100	84	128	84	100	84	126	82	100	84	128	82	100	84	128	82	100	82	128	80	100	80	126	80	100	82	126	80	99
21	96	134	90	100	82	132	82	100	84	128	82	100	82	126	82	100	82	126	82	100	82	128	82	100	82	126	82	100	82	126	80	100
22	92	128	88	99	82	130	84	99	84	130	82	100	84	128	84	99	82	128	82	99	84	126	82	99	82	126	82	100	82	126	82	100
23	94	130	90	100	86	128	86	100	82	126	84	100	84	126	84	100	84	126	82	99	82	128	82	100	82	126	82	99	82	126	82	100
24	96	132	92	99	88	130	82	100	86	128	86	100	82	128	82	100	84	128	80	99	82	126	82	100	82	126	80	100	82	126	82	100
25	95	134	94	100	82	132	88	100	82	130	84	99	84	128	84	100	82	126	82	100	80	128	80	100	82	128	80	100	80	128	80	99

GROUP - RD (Ropivacaine with Dexmedetomidine)

S. No		0 min					15 mins				30 mins				60 min				120 mins				180 mins				240 min				300 mins		
	HR	SBP	DBP	SPO2	HR	SBP	DBP	SPO2	HR	SBP	DBP	SPO2	HR	SBP	DBP	SPO2	HR	SBP	DBP	SPO2	HR	SBP	DBP	SPO2	HR	SBP	DBP	SPO2	HR	SBP	DBP	SPO2
1	94	136	90	100	82	126	80	100	76	110	70	100	70	108	68	99	66	108	68	100	66	106	68	100	64	106	68	99	66	108	66	100
2	92	134	90	100	84	124	78	100	74	110	68	100	70	108	68	100	68	106	68	99	66	106	68	100	64	106	68	100	66	108	66	100
3	92	132	92	100	82	126	78	99	74	110	70	100	70	108	68	100	66	106	68	100	66	106	68	100	66	106	68	100	66	108	66	100
4	92	130	90	100	76	128	78	100	74	110	70	99	70	108	68	99	68	106	68	100	64	106	68	99	66	106	68	100	66	108	68	100
5	93	130	92	100	80	128	78	99	74	108	70	100	68	106	68	100	66	106	68	100	64	106	68	100	66	106	66	100	66	108	66	100
6	91	128	92	99	80	126	76	100	70	108	68	100	68	106	66	100	66	106	66	100	64	106	68	100	66	108	68	100	66	108	66	100
7	92	130	94	100	78	124	76	100	70	108	68	100	68	106	66	99	66	106	66	100	64	106	66	100	66	108	68	100	66	108	66	100
8	96	134	90	100	78	124	76	100	70	108	68	100	68	106	66	100	58	106	66	100	62	106	66	100	62	108	68	100	66	110	70	99
9	95	132	92	100	78	124	76	99	70	108	68	99	68	104	66	100	66	104	66	100	62	108	66	100	62	108	68	100	64	108	68	100
10	93	136	88	99	78	124	76	100	70	108	68	100	66	106	66	100	66	104	66	100	62	108	66	100	62	108	68	100	64	108	68	100
11	94	128	82	100	76	124	74	100	68	110	70	100	66	106	66	100	66	104	66	100	62	108	66	100	62	108	68	100	64	108	68	100
12	91	130	80	100	76	128	76	99	68	110	70	100	66	106	66	100	64	104	66	100	62	108	64	100	60	108	68	100	64	108	68	100
13	95	134	82	100	76	126	74	100	68	108	70	100	66	106	66	100	64	104	66	100	60	106	64	100	60	106	66	100	64	108	68	100
14	95	128	80	100	74	126	74	100	68	106	70	99	66	106	68	100	64	104	66	100	62	106	64	100	60	106	66	100	64	110	70	100
15	93	130	86	100	74	126	74	100	68	106	68	100	66	104	66	99	64	104	66	100	62	106	64	100	60	106	66	100	64	108	68	100
16	92	132	86	100	74	126	74	100	70	106	68	100	64	104	66	100	58	104	66	100	62	106	64	100	60	106	66	100	64	108	68	100
17	91	134	88	100	74	126	74	99	70	106	68	100	64	104	66	100	64	104	66	100	62	106	66	100	60	106	68	100	62	108	68	100
18	92	130	90	99	74	124	72	100	70	108	68	100	64	104	66	100	64	104	66	100	62	106	66	100	60	106	68	100	64	108	68	100
19	93	132	92	100	74	122	72	100	70	108	66	100	64	104	66	100	64	104	66	100	62	106	60	99	60	106	68	100	64	108	68	99
20	95	130	88	100	72	128	72	100	70	108	68	100	64	106	66	100	62	102	64	100	64	106	60	100	62	108	68	100	64	108	68	100
21	94	134	90	100	72	126	74	100	68	108	68	100	64	106	66	100	62	106	64	100	62	104	64	100	62	108	66	100	64	108	68	100
22	94	128	88	99	72	126	72	100	68	106	68	100	66	106	66	100	62	106	64	99	62	104	64	100	62	108	66	100	64	108	68	100
23	95	130	90	100	72	128	72	100	68	106	68	100	66	106	68	100	62	106	64	100	62	104	64	100	62	108	66	100	64	110	68	100
24	96	132	90	100	76	120	72	100	68	106	68	100	66	106	68	100	62	106	64	99	62	104	64	100	60	108	66	100	64	108	68	100
25	95	134	94	100	82	124	70	100	66	106	66	99	64	106	66	100	60	104	64	100	60	106	64	100	60	106	66	99	62	108	66	100

GROUP - R (Ropivacaine)

					Minutes							VAS					Sed.scores			
S. No	Age	Sex	Wt.	ASA	DOS	On.sen	On.mot	DO.sen	DO.mot	DOA	Res. An.	6 h	10 h	14 h	18 h	24 h	1 hr	2 hr	3 hr	S/E
1	25	f	70	II	110	9	12	480	460	560	580	0	4	6	8	10	1	1	1	nil
2	28	f	74	I	170	10	13	495	475	580	600	1	5	6	8	10	1	1	1	nil
3	26	m	78	I	130	8	11	510	485	570	580	0	5	7	9	10	1	1	1	nil
4	30	f	68	I	190	10	14	525	500	570	590	0	4	6	8	10	1	1	1	nil
5	32	m	64	I	170	8	14	470	450	550	570	0	4	7	9	10	1	1	1	nil
6	34	f	66	I	160	10	14	490	460	570	590	0	4	6	8	10	1	1	1	nil
7	36	m	62	I	190	9	13	515	485	560	580	0	4	6	8	10	1	1	1	nil
8	52	m	62	I	170	9	13	520	495	580	600	1	5	7	9	10	1	1	1	nil
9	45	m	67	II	170	9	13	535	500	580	600	1	5	6	8	10	1	1	1	nil
10	38	m	68	I	140	8	12	485	445	570	590	0	4	6	8	10	1	1	1	nil
11	24	f	64	I	150	10	14	490	470	580	600	0	4	6	8	10	1	1	1	nil
12	26	f	68	I	160	10	14	505	485	580	600	0	4	6	9	10	1	1	1	nil
13	28	m	70	I	180	8	12	515	495	570	590	0	4	7	9	10	1	1	1	nil
14	30	f	72	I	190	9	12	520	495	600	620	1	5	6	8	10	1	1	1	nil
15	32	f	74	I	160	9	13	535	500	600	615	1	5	6	8	10	1	1	1	nil
16	53	m	68	I	170	9	13	540	520	580	595	0	4	7	9	10	1	1	1	nil
17	46	m	70	I	180	10	14	515	495	540	560	0	4	7	9	10	1	1	1	nil
18	42	m	72	II	160	10	14	485	450	550	575	0	4	6	8	10	1	1	1	nil
19	28	m	77	I	180	8	12	490	470	550	575	0	5	7	9	10	1	1	1	nil
20	24	m	76	I	150	9	13	510	480	570	590	0	4	6	8	10	1	1	1	nil
21	26	m	69	I	140	10	14	505	475	560	580	0	4	6	8	10	1	1	1	nil
22	28	f	68	I	150	10	14	500	470	560	580	0	4	7	8	10	1	1	1	nil
23	34	m	70	I	160	9	13	490	460	550	570	0	5	7	9	10	1	1	1	nil
24	38	m	68	I	170	9	13	505	470	545	570	0	4	6	8	10	1	1	1	nil
25	36	m	72	I	180	10	14	525	480	580	600	1	5	6	8	10	1	1	1	nil

GROUP - RD (Ropivacaine with Dexmedetomidine)

					Minutes							VAS					Sed. scores			
S. No	Age	Sex	Wt.	ASA	DOS	On.sen	On.mot	DO.sen	DO.mot	DOA	Res. An.	6 h	10 h	14 h	18 h	24 h	1 hr	2 hr	3 hr	S/E
1	26	m	70	II	120	7	10	680	665	885	905	0	0	4	6	8	2	2	2	nil
2	27	f	74	I	180	7	10	710	680	850	870	0	0	4	6	8	2	2	2	nil
3	25	m	78	I	130	6	9	690	675	890	910	0	0	5	7	10	2	2	2	nil
4	29	f	68	I	180	6	9	720	695	840	860	0	0	4	6	8	2	2	2	nil
5	31	m	64	I	180	7	9	670	645	880	900	0	0	4	6	8	2	2	2	nil
6	32	f	66	I	170	6	8	695	670	825	840	0	0	4	6	8	2	2	2	nil
7	36	m	60	I	170	7	9	700	675	815	840	0	0	4	6	8	2	2	2	nil
8	54	m	62	I	180	7	9	685	635	875	895	0	0	4	6	8	3	3	3	nil
9	44	m	64	II	170	7	9	685	660	890	910	0	0	5	7	10	2	2	2	nil
10	39	m	68	I	150	7	10	690	665	795	810	0	1	4	6	8	2	2	2	nil
11	25	f	64	I	150	6	8	725	675	810	825	0	0	4	6	8	2	2	2	nil
12	25	f	68	I	160	6	8	760	680	815	830	0	0	4	6	8	3	3	3	nil
13	29	m	70	I	170	7	9	740	695	825	840	0	0	4	6	8	2	2	2	nil
14	29	m	72	I	180	7	9	680	655	795	810	0	0	4	6	8	2	2	2	nil
15	31	f	74	I	160	7	9	695	670	805	825	0	0	4	6	8	3	3	3	nil
16	54	m	68	II	160	7	9	785	685	815	840	0	0	4	6	8	2	2	2	nil
17	45	m	70	I	180	8	10	785	655	825	840	0	0	4	5	8	2	2	2	nil
18	43	m	72	II	150	8	10	720	690	825	840	0	0	4	5	8	2	2	2	nil
19	29	m	74	I	170	6	9	695	655	825	845	0	0	4	5	8	2	2	2	nil
20	25	m	76	I	160	7	9	745	645	890	910	0	0	5	6	8	2	2	2	nil
21	27	m	69	I	150	7	9	680	660	815	840	0	0	4	6	8	2	2	2	nil
22	29	f	68	I	140	7	10	670	695	835	845	0	0	4	6	8	2	2	2	nil
23	33	m	70	I	160	6	8	695	645	790	810	0	0	4	6	8	2	2	2	nil
24	39	m	68	I	160	6	8	740	720	790	810	0	1	4	6	8	2	2	2	nil
25	37	m	72	I	170	8	8	685	640	790	810	0	2	4	6	10	2	2	2	nil

INSTITUTIONAL ETHICAL COMMITTEE
GOVT.KILPAUK MEDICAL COLLEGE,
CHENNAI-10
Ref.No.3182/ME-1/Ethics/2014 Dt:08.05.2014.
CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval "A Comparative Study of Ropivacaine 0.5 and Ropivacaine 0.5% with dexmedetomidine 50µg in ultrasound guided supraclavicular brachial plexus block for upper limb orthopaedic surgery" – For Project Work.submitted by Dr.H.M.Hajashareef, MD (Anaes), PG Student, KMC, Chennai-10

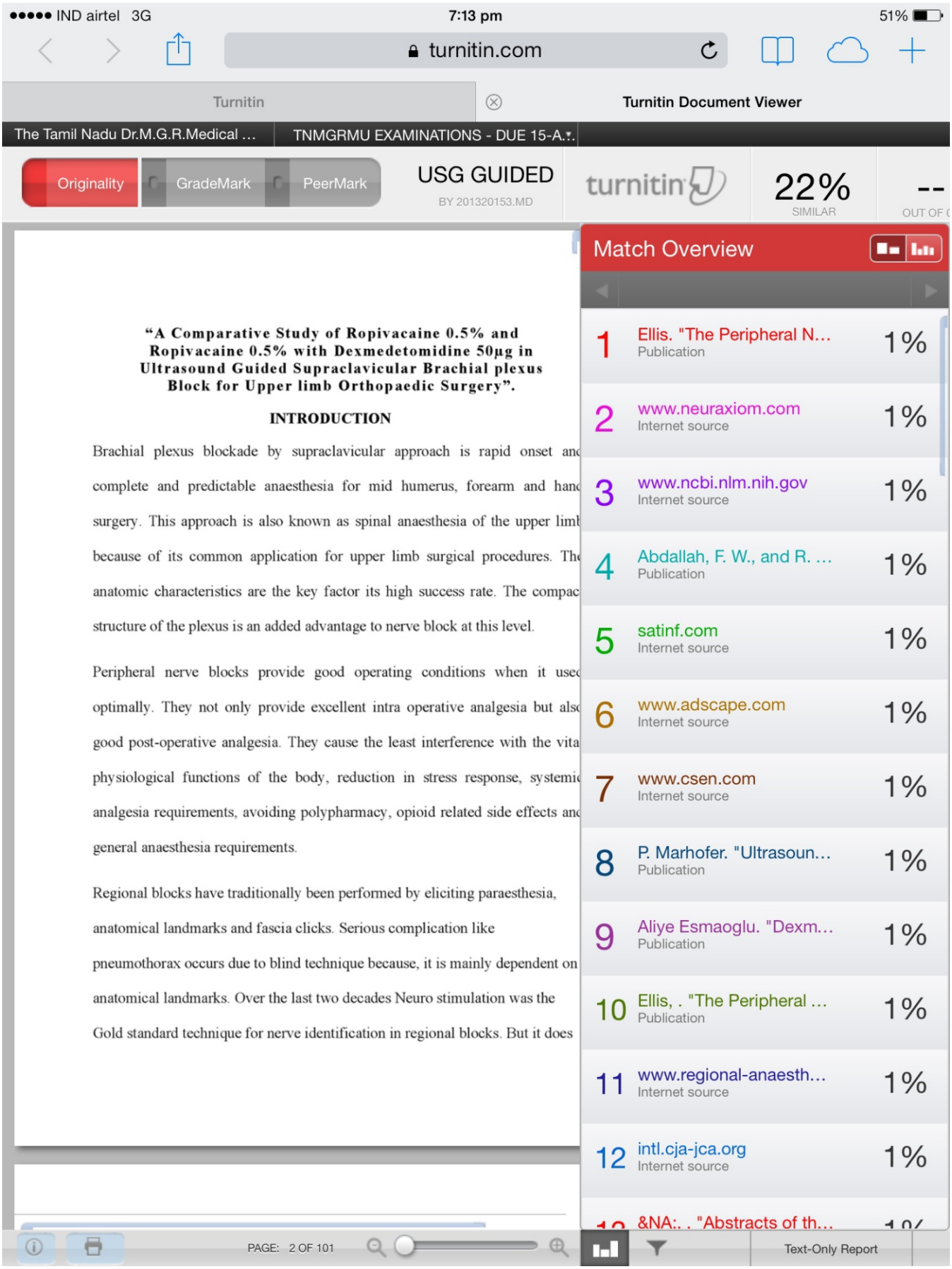
The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

30/5/14.

CHAIRMAN,
Ethical Committee
Govt.Kilpauk Medical College,Chennai

GOVT. KILPAUK MEDICAL COLLEGE
30 MAY 2014
Kilpauk, Chennai-10.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OUT OF
"A Comparative Study of Ropivacaine 0.5% and Ropivacaine 0.5% with Dexmedetomidine 50µg in Ultrasound Guided Supraclavicular Brachial plexus Block for Upper limb Orthopaedic Surgery".
INTRODUCTION
Brachial plexus blockade by supraclavicular approach is rapid onset and complete and predictable anaesthesia for mid humerus, forearm and hand surgery. This approach is also known as spinal anaesthesia of the upper limb because of its common application for upper limb surgical procedures. The anatomic characteristics are the key factor its high success rate. The compact structure of the plexus is an added advantage to nerve block at this level.
Peripheral nerve blocks provide good operating conditions when it used optimally. They not only provide excellent intra operative analgesia but also good post-operative analgesia. They cause the least interference with the vital physiological functions of the body, reduction in stress response, systemic analgesia requirements, avoiding polypharmacy, opioid related side effects and general anaesthesia requirements.
Regional blocks have traditionally been performed by eliciting paraesthesia, anatomical landmarks and fascia clicks. Serious complication like pneumothorax occurs due to blind technique because, it is mainly dependent on anatomical landmarks. Over the last two decades Neuro stimulation was the Gold standard technique for nerve identification in regional blocks. But it does
Match Overview
1 Ellis. "The Peripheral N... Publication 1%
2 www.neuraxiom.com Internet source 1%
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4 Abdallah, F. W., and R. ... Publication 1%
5 satinf.com Internet source 1%
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PAGE: 2 OF 101
Text-Only Report

PATIENT INFORMED CONSENT FORM

Study Detail : "A Comparative Study of Ropivacaine 0.5% and Ropivacaine 0.5% With Dexmedetomidine 50µg in Ultrasound Guided Supraclavicular Brachial plexus Block for Upper limb Orthopedic Surgery".

Study Centre : GOVT. KILPAUK MEDICAL COLLEGE & HOSPITAL, CHENNAI.

Patient's Name :

Patient's Age :

Identification Number :

Patient may check ( ) these boxes

I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction. ☐

I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected. ☐

I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well-being or any unexpected or unusual symptoms. ☐

I hereby consent to participate in this study. ☐

I hereby give permission to undergo complete clinical examination and diagnostic tests including hematological, biochemical, radiological tests. ☐

Signature/thumb impression:

Patients Name and Address : Place: Date:

Signature of investigator :

Study investigator's Name : Place: Date:

Consent form in regional language (Tamil)

ஒப்புதல் படிவம்

ஆய்வு செய்யப்படும் தலைப்பு :

துறை :

கீழ்ப்பாக்கம் மருத்துவ கல்லூரி :

பங்கு பெறுபவரின் பெயர் :

பங்கு பெறுபவரின் வயது :

பங்கு பெறுபவரின் எண் :

மேலே குறிப்பிடப்பட்டுள்ள ஆராய்ச்சியின் விவரங்களும் அதன் நோக்கமும் முழுமையாக எனக்கு தெளிவாக விளக்கப்பட்டது.

எனக்கு விளக்கப்பட்ட விஷயங்களை நான் புரிந்து கொண்டு எனது சம்மதத்தை தெரிவிக்கிறேன்.

இந்த ஆராச்சியில் பிறரின் நிர்ப்பந்தமின்றி என் சொந்த விருப்பத்தின் பேரில் தான் பங்கு பெறுகின்றேன். இந்த ஆராய்ச்சியில் இருந்து நான் எந்நேரமும் பின்வாங்கலாம் என்பதையும் அதனால் எந்த பாதிப்பும் ஏற்படாது என்பதையும் நான் புரிந்து கொண்டேன்.

நான் என்னுடைய சுய நினைவுடனும் மற்றும் முழு சுதந்திரத்துடனும் இந்த மருத்துவ ஆராய்ச்சில் என்னை சேர்த்துக் கொள்ள சம்மதிக்கிறேன்.

ஆராய்ச்சியாளர் மற்றும் அவரைச் சார்ந்தவர்களோ, நெறிமுறைக்குழு உறுப்பினர்களோ நான் இந்த ஆராய்ச்சியில் இருந்து விலகினாலும் என்னுடைய அனுமதியின்றி என்னுடைய உடல்நிலை குறித்த தகவல்களை இந்த ஆராய்ச்சிக்கோ இது தொடர்பான வேறு ஆராய்ச்சிக்கோ பயன்படுத்திக் கொள்ள முடியும் என்று புரிந்து கொண்டு சம்மதம் அளிக்கிறேன். ஆனாலும் என்னுடைய அடையாளம் வெளியிடப்பட மாட்டாது என்று புரிந்து கொள்கிறேன்.

இந்த ஆராய்ச்சியின் தகவல்களையும் முடிவுகளையும் அறிவியல் நோக்கத்திற்காக பயன்படுவதற்கு நான் அனுமதியளிக்கிறேன். இந்த ஆராய்ச்சியில் பங்கு பெற முழு மனதுடன் சம்மதிக்கிறேன்.

ஆய்வாளர் பெயர்
மற்றும் கையொப்பம்

பங்கேற்பவரின் கையொப்பம்
(அல்லது) கட்டை விரல் ரேகை